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A CONTRIBUTION TO THE PATHOLOGY OF PARALYSIS AGITANS*

CHARLES METCALFE BYRNES, M.D.

BALTIMORE

From the time of Parkinson to within the past decade, paralysis agitans has been attributed to a variety of lesions, from minor cytologic changes to gross anatomic destruction, in numerous and widely separated portions of the central nervous system. Examination of the peripheral nerves had failed to add material knowledge of a positive nature. The internal secretory glands and the striated musculature were also studied without furnishing any convincing pathologic evidence. In 1911, Mendel[1] published a comprehensive account of the disease with a critical review of the pathologic studies recorded up to that period. Evidence was accumulating which suggested that the lesion might be situated in one or more of the central nuclear masses. Brissaud, Maillard and Jelgersma had observed changes in the substantia nigra, in the red nucleus, and in the lenticulostriate and hypothalamic structures; but the lesions were often diffuse, wide-spread, and of uncertain meaning. Similar observations were recorded later by Levy, Monschat, and Auer and McCough. In 1918, Hunt directed special attention to structural changes in the large cells of the globus pallidus and putamen, with loss of fibers in the striothalamic region; these, he thought, were the essential lesion in a case of juvenile paralysis agitans. The subthalamic nuclei were said to be unaltered. Suggestive as they were, these studies were discredited by repeated instances in which anatomic examination failed to reveal any change in the central nervous tissue. Paralysis agitans remained the "sphinx" of neurology.

Clinically, the disease is equally obscure. It has been thought that the frequent unilateral appearance of the symptoms, the cessation of the tremor and rigidity during sleep and following cerebral hemorrhage, the subjective sensory disorders and the psychic manifestations are difficult of explanation on any other basis than that of a central lesion. Con-

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* Presidential address: read in abstract before the Philadelphia Neurological Society, Jan. 25, 1924.

* From the Laboratory of Neuropathology, the Church Home and Infirmary, which provided the laboratory facilities necessary for the completion of this study.

1. Mendel, K.: Die Paralysis agitans, Berlin, S. Karger, 1911.

tradictory clinical evidence, however, is not wanting. The affection is often bilateral from the outset; psychic disorders are commonly absent or occur only late in the course of the disease, and the motor features are not always abolished during sleep or after capsular lesions. In fact, Parkinson [2] observed that, "tremulous motions of the limbs occur during sleep and augment until they waken the patient."

Notwithstanding the prevailing opinion as to the cerebral location of the lesion and the essentially nervous character of the affection, it was apparent that, for a period of one hundred years, searching study of the nervous system had failed to disclose a definite pathologic foundation for the disease, and that the muscle tissue, the most conspicuous anatomic structure concerned in the mechanism of motion, had not been thoroughly investigated. Although the myopathic nature of the disease had been discredited, the occasional early involvement of the small muscles of the hand, and the fact that there are in the skeletal musculature a variety of nerve terminals which had not been carefully examined, suggested that the somatic neuromuscular tissue might be profitably studied.

The postencephalitic parkinsonian syndrome with a much disfigured lenticulostriate body disturbed, for a time, my belief that the lesion was to be found outside the central nervous system. Clinicians and pathologists pursued with renewed interest the study of this strange likeness of Parkinson's disease. Opportunities for anatomic investigation were soon forthcoming, but once more the lesions were of varied type; they were not uniformly distributed or confined to any one of the central nuclei, and occasional contradictory evidence was obtained. Thus, while much of the recent knowledge of the pathology of the disease has been derived from a study of the postencephalitic affection, material from cases of genuine paralysis agitans has also been subjected to equally searching investigation, without contributing any additional evidence as to the exact location of the lesion. Nevertheless, it appears that the parkinsonian syndrome, and particularly the encephalitic form, is often associated with disease of one or more of the following structures: the globus pallidus, the substantia nigra, the hypothalamic nuclei, the thalamus, the putamen, the caudate nucleus, the ansa lenticularis and the frontopontocerebellar system of fibers; but destructive lesions within this ganglionic area are also accompanied by a number of different types of motor phenomena which are designated as various other clinical entities.

In a general review of the subject by Souques,[3] it is asserted that all efforts to establish the precise location of the lesion in either the

2. Parkinson, J.: An Essay on the Shaking Palsy, London, Sherwood, Neely and Jones, 1817.

3. Souques, M. A.: Rapport sur les syndromes parkinsoniens, Rev. neurol. **37**:534 (June) 1921.

encephalitic or the genuine parkinsonian syndrome have been unsuccessful, but that three current opinions are held as to the probable situation of the morbid process. According to Hunt the lesion is confined to the large motor cells of the pallidal system. Trétiakoff, although contradicted in the studies of Hunt, Jelgersma, Levy and the Vogts, attributes the disease to a lesion in the substantia nigra, while S. A. K. Wilson is of the opinion that the essential change is situated in the red nucleus and in the corpus Luysi. Recently, McKinley [4] has noted degeneration of the cells in the substantia nigra, although the lesion was not confined to this nucleus; and it is particularly significant that Lhermitte and Cornil found disease of the substantia nigra without evidences of the parkinsonian syndrome. Disintegration of the large cells in the caudate and lenticular nuclei, unaccompanied by any of the parkinsonian symptoms, has likewise been noted by Guillain and P. Marie; and Wilson's localization of the lesion in the red and subthalamic nuclei has also met with contradictory evidence.

Not only does this uncertainty exist in regard to the location of the pathologic process, but there is also no unanimity of opinion concerning the clinical identity of the postencephalitic and the genuine parkinsonian syndrome. Both are unquestionably characterized by tremor, hypertonia and loss of associated and automatic movements, but very often the two are distinguishable: in the encephalitic type by the history of a preceding encephalitis, the onset and development of the symptoms, the predominance of rigidity and vasomotor and secretory disorders; in the parkinsonian disease in the character of the tremor with its occasional restriction for prolonged periods to the fingers of one or both hands.

Important knowledge has, however, been added to the anatomy and physiology of this central nuclear area, and its pathology cannot be ignored in the study of these various disorders of muscle tone. So far as paralysis agitans is concerned, Souques makes an agreeable summary of the pathologic data when he states that the lesion is presumably situated in the striatum or the pallidum although it cannot be located with precision. It may be characterized by a variety of changes, gross or microscopic, affecting the corpus striatum, the substantia nigra or the subthalamic region. Oppenheim,[5] likewise, is unable to arrive at a more definite conclusion than that the evidence seems to indicate a lesion somewhere within the basal ganglia.

This indefinite and contradictory state of knowledge regarding the pathology of paralysis agitans encouraged my belief that further infor-

4. McKinley, J. C.: Lesions in the Brain of a Patient with Postencephalitic Paralysis Agitans, Arch. Neurol. & Psychiat. **9**:47 (Jan.) 1923.

5. Oppenheim, H.: Lehrbuch der Nervenkrankheiten, Ed. 7, Berlin, S. Karger, **2**:2069, 1923.

mation might be derived from a study of the muscular tissue and its proprioceptive system of nerve terminals; and it is to a lesion in this system that attention is here directed.

THE MUSCLE SPINDLE

Distribution of the Spindle.—Terminating in the striated musculature there are five types of nerve-endings: the motor end-plate, the pacinian corpuscle, the neurotendinous end-organ of Golgi, the sympathetic non-medullated axon and the intricate network of fibers peculiar to the muscle spindle. With the exception of the motor fibers and those from the sympathetic system, all of these axons conduct afferent impulses to the central nervous system through the posterior rami of the spinal nerves; but, unlike the efferent fibers, these axons have no direct ending in the muscle tissue proper.

It is scarcely conceivable that all the skeletal muscles have been searched for the presence of spindles, but it is stated that they make their appearance during the fourth month of fetal life and are found throughout the striated musculature, with the exception of the extra-ocular muscles, the diaphragm and the intrinsic muscles of the tongue. Although Sutton [6] has demonstrated them in the extrinsic eye muscles of the embryo pig, Sherrington was unable to find them in these muscles in the cat or in the monkey; and Batten states that they do not occur in the extra-ocular muscles of the human being. In a series of transverse sections through the four rectus muscles of the human eye I was unsuccessful in demonstrating spindles, although Siemerling claims to have found them in the adult human rectus oculi which had suffered great atrophic changes. Langworthy [7] has recently studied the spindle in the extrinsic tongue musculature of the cat, but was unable to demonstrate them in the intrinsic musculature. More exact information concerning the spindle content of these muscles is much needed, since it is maintained that the diaphragm, the intrinsic muscle of the tongue and the extra-ocular muscles are not affected in genuine paralysis agitans. Ordenstein [8] has, however, described involvement of the extra-ocular muscles in advanced stages of the disease.

Although the spindles are widely distributed, it is noteworthy that they are particularly abundant in the small muscles of the hands and feet, in which they are readily obtained in specimens secured from the

6. Sutton, A. C.: On the Development of the Neuromuscular Spindle in the Extrinsic Eye Muscles of the Pig, Am. J. Anat. **18**:117, 1915.

7. Langworthy, O. R.: A Study of the Innervation of the Tongue Musculature with Particular Reference to the Proprioceptive Mechanism, J. Comp. Neurol. **36**:273 (Feb.) 1924.

8. Ordenstein, L.: Paralysie agitante et la sclérose en plaques généralisée, Thèse de Paris, 1867.

thenar, hypothenar and plantar muscles. I have found as many as twenty spindles in a specimen of muscle measuring 3.5 by 1 cm. obtained from the abductor pollicis brevis, whereas the biceps brachii is said to contain not more than seventy. Sherrington states that they are also especially numerous just beneath the aponeurosis at the distal end of the vastus medialis, and he finds that if this aponeurosis is removed, no matter how carefully, the knee jerk is always abolished.

Structure of the Spindle.—Situated, then, within most of the striated musculature are these elongated, spindle-shaped bodies lying at times obliquely but, as a rule, parallel to the course of the surrounding muscle fibers. Because of the early uncertainty concerning their nature and function, they were designated by various names and, since they were more readily discovered in the atrophic muscle, they were regarded as pathologic formations, as muscle in the process of division and as centers of regenerating muscle. They were accordingly designated by Kölliker as "Muskelknospen" or musclebuds, but Kühne,[9] who also considered them centers of growth, was the first to speak of them as "die Muskelspindeln." Babinski [10] claims to be the first to recognize the spindle in man, although he was, according to Spiller, preceded by Roth of Moscow, who observed a similar structure in the human muscle and described it as the neuromuscular bundle. Further knowledge of the history and anatomy of the spindle is found in the writings of Laura Forster [11] and in a later monograph by Batten,[12] in 1897, during which year the spindle appears to have been an object of special investigation, since additional studies then appeared by Huber and De Witt,[13] Ruffini,[14] Horsley, Grünbaum and Spiller.

The spindle, as the name implies, is a fusiform body consisting of a central bundle of specially constructed muscle fibers, an intricate nerve plexus and intrafusal blood vessels. It is enclosed in a white fibrous connective tissue capsule which attains its greatest width in the equatorial

9. Kühne, W.: Die Muskelspindeln, Virchows Arch. f. path. Anat. **28**:528, 1863.

10. Babinski, J.: Sur la présence dans les muscles striés de l'homme d'un système spécial constitué par des groupes de petites fibres musculaires entourées d'une gaine lamelleuse, Soc. de Biol. Paris **38**:629, 1886; also, Rev. de méd., 1884, p. 246.

11. Forster, Laura: Zur Kentniss der Muskelspindeln, Virchows Arch. f. path. Anat. **137**:121, 1894.

12. Batten, F. E.: The Muscle Spindle Under Pathological Conditions, Brain **20**:138, 1897.

13. Huber, G. C., and De Witt, L. M. A.: A Contribution on the Motor Nerve Endings and on the Nerve Endings in the Muscle Spindle, J. Comp. Neurol. **7** and **8**, 1897-1898.

14. Ruffini, A.: Observations on Sensory Nerve-Endings in Voluntary Muscles, Brain **20**:368, 1897.

region and tapers into a proximal and a distal polar extremity at which it fuses with the surrounding endomysium. When the spindle is situated near the attachment of the muscle, one pole of the capsule often terminates in the tendinous insertion.

Ordinarily, the spindle is larger in the adult than in the child and, according to Batten, attains its greatest dimensions in the long muscles of the extremities, where it has a usual length of from 2 to 4 mm., and sometimes measures as much as 11 mm. The equatorial width varies from 0.15 to 0.5 mm. Thus, the teased, isolated spindle may be easily recognized with the unaided eye. More accurate measurements of its transverse dimensions are obtained, however, from a study of cross-sections of embedded material, and by this means Batten, Horsley,[15] Babinski, and Sherrington[16] found the greatest equatorial diameter not more than 0.240 mm.; but they failed to state whether or not this measurement included the capsule.

Occasionally, two spindles are united at their polar extremities, or one may be superimposed on the other; although this compound arrangement is not uncommon, Batten states that he has never seen more than two spindles thus united. Sherrington, however, has observed three, and Kerschner claims to have found four specimens partly enclosed in a common sheath, although each spindle retained its own capsule and separate innervation. Sometimes two, three, or even four spindles are in end-to-end contact without being actually united.

Each neuromuscular bundle possesses a distinct capsule in which the lamellae of white fibrous connective tissue with elongated nuclei are arranged in an onion-like fashion. The lamellae vary in number from six to eight and are either closely applied to one another or separated by a narrow space devoid of any visible material. Although the nuclei and the capsular layers are quite apparent in the teased spindle, the minute structure of the capsule and its relation to the enclosed structures are more readily distinguished in microscopic sections.

In a properly stained and teased specimen the intrafusal muscle is apparent as a dark bundle running longitudinally throughout the length of the spindle, although with the unaided eye or with low magnification it is impossible to distinguish the individual muscle fibers. These are of the red variety and are said to enter the spindle at its proximal pole. The cross and longitudinal striations are coarser than those of the extrafusal muscle and, although especially conspicuous in the polar

15. Horsley, Sir V.: Short Note on the Sense Organs in Muscle and on the Preservation of Muscle Spindles in Conditions of Extreme Muscular Atrophy Following Section of the Motor Nerve, Brain **20**:375, 1897.

16. Sherrington, C. S.: On the Anatomical Constitution of Nerves of Skeletal Muscles with Remarks on Recurrent Fibres in the Ventral Spinal Nerveroots, J. Physiol. **17**:211, 1894-1895.

regions, are often indistinct or absent at the equator. Occasionally, the striations are apparent throughout the length of the fiber. The single spindle contains from two to ten muscle fibers and as many as twenty have been found in the compound spindle. These are ordinarily arranged parallel to each other, but sometimes they are contorted and irregularly disposed. In the equatorial region the muscular bundle is usually placed excentrically, and is much darker in color than elsewhere since in this region it is surrounded by a mass of spherical or ovoid nuclei measuring 0.006 mm. in diameter. This nuclear material is also embedded within the muscle cells and has a longitudinal extent of approximately 0.400 mm.

The intrafusal muscle fibers vary in size and, ordinarily, are larger at the poles than at the equator where the parent fibers undergo longitudinal division. They are also smaller, of different structure and more nearly circular than those of the surrounding muscle; and, according to Huber, they may or may not possess a sarcolemma sheath. Batten states that they have an average diameter of 0.020 mm., but may be as small as 0.008 mm. at the equator, whereas the extrafusal fibers are usually not less than 0.060 mm. in diameter. Kerschner has described a peculiar type of normal intrafusal muscle which has apparently been regarded by others as a pathologic formation. He observed at the periphery of the muscle bundle a chain of elongated cells with oval nuclei and a faintly striated cytoplasm. These muscle cells, situated just beneath the axial sheath, often extend through the length of the spindle, and are not so large or so deeply stained as the more centrally placed fibers. I, also, have found this type of cell in the normal spindle, as well as the central core of hyaloid material sometimes apparent in equatorial cross-section of the muscle. This central core is surrounded by a ring of fibrillated sarcoplasm at the periphery of which is a nuclear layer derived from the adjacent connective tissue.

The entire muscle bundle is surrounded by a closely applied axial sheath, similar in structure to the capsule, but separated from it by a space of variable width known as the periaxial space. According to Sherrington, the width of the periaxial space varies in the polar region from 0.005 to 0.015 mm., and from 0.040 to 0.060 mm. at the equator. The space is crossed at intervals by a very delicate network of connective tissue extending from the axial sheath to the inmost layer of the capsule, but ordinarily it contains no other formed elements. Sometimes the space is very irregular in outline because of invagination or evagination of the capsule from contraction of the extrafusal musculature; and it is said to contain, at times, a small amount of amorphous precipitated proteid or an occasional lymphocyte, but never any red blood cells. Sherrington demonstrated the lymphatic nature of the periaxial space

when he injected it successfully in the spindles of the intrinsic plantar muscles, with a colored solution introduced into the lymphatic vessels of the leg.

Each spindle is innervated by at least two nerve trunks, one of which usually enters the capsule at the equator, while the other is directed to one of the polar regions. Huber found that each nerve to the smaller spindle contains from one to four medullated fibers, whereas those to the larger and, especially, to the compound spindle convey as many as eight. After piercing the capsule, the nerve crosses the periaxial space, encircles the muscular bundle and enters the axial sheath, in which it loses its medullary covering, and terminates, finally, in various fashions on the intrafusal muscle. As the nerve approaches the capsule, the internodal segments become shorter; this reduction in length continues as the nerve traverses the periaxial space.

The innervation of each spindle is in all probability specific. Although two or three spindles may be joined together, Sherrington states that he has never seen a nerve fiber passing from one to the other, and my own dissections confirm this observation. It is claimed by Kerschner, however, that the neurotendinous end-organ and the muscle spindle are innervated by a single nerve; but Catanneo, according to Batten, observed a spindle lying between two neurotendinous end-organs, and demonstrated, in this instance, their separate innervation. In the material from one of my cases I found, at the terminal portion of a single nerve trunk, a spindle, a neurotendinous end-organ and two pacinian corpuscles; each of these structures possessed its own nerve trunk, however, and there was no evidence that they received a common innervation.

There is much discussion concerning the presence of motor fibers in the neuromuscular bundle. Huber and Kerschner both claim to have demonstrated motor end-plates in at least some of the spindles, but Sherrington was unable to discover this type of nerve ending and, while he did not regard this as proof of its absence, he was of the opinion that the spindle receives no intrinsic motor axons. Batten, likewise, was unsuccessful in demonstrating true motor terminals in any of his specimens. The nerve endings in the spindle have been divided by Ruffini into three types: the looplike, the flower or wreathlike and the platelike ending which resembles closely the motor endplates; and, no doubt, it is this third type about which a diversity of opinion has been expressed. This is in all probability the type observed in some of my specimens and mistaken by others, as well as by me, for a true motor nerve terminal. Notwithstanding the uncertainty as to the nature of this third type of ending, there is convincing experimental and pathologic evidence that the spindle receives no direct innervation from the lower motor neuron.

Anatomists and clinicians are now much interested in the sympathetic innervation of the striated musculature, and it would be instructive to know whether or not the spindle receives any axons from this system. Dart [17] has recently demonstrated, in the neuromuscular bundle, nerve terminals with a disposition and configuration similar to those of the sympathetic terminals in the surrounding muscle. These, he believes, are the endings of autonomic fibers; and, although this system of fibers may contain a small number of very fine medullated axons, Dart found no typical nonmedullated axons within the spindle. Nevertheless, this suggestive histologic evidence finds no confirmation in the experiments of Sherrington by whom it was demonstrated that if both the anterior and the posterior roots were sectioned, all nerves to the spindle and to the neurotendinous end-organ underwent complete degeneration. He therefore concluded that neither of these bodies receives any direct innervation from the autonomic nervous system. It is conceivable, however, that sympathetic fibers might be conveyed to the muscle bundle by means of the blood vessels, but Langworthy found all nerve fibers to the spindle completely degenerated after section of the peripheral trunk, although small nonmedullated axons were preserved along the course of the blood vessels in the surrounding muscle.

Function of the Spindle.—Intimations of its function are derived from the peculiar circumstances under which the spindle is most readily demonstrated. It is commonly observed that the spindle is especially conspicuous in the atrophic muscle, and that in disease of the lower motor neuron its integrity is preserved when the surrounding muscle tissue is totally degenerated.

This indication that the spindle receives no innervation from the lower motor neuron suggested that its nerve fibers were derived from the posterior root ganglion, and Mays, Kerschner and Ruffini accordingly regarded the spindle as a sensory end-organ. Sherrington furnished experimental evidence in support of this opinion when he cut all the motor fibers to a muscle and, after allowing an interval of time sufficient for degenerative changes, found the motor nerve terminals completely disintegrated and the muscle greatly atrophied; but the nerves in the neuromuscular bundle were unaltered. In a second experiment the posterior roots alone were sectioned distal to the ganglion and after all conditions of the experiment were fulfilled, degenerative changes were noted in the nerve fibers of the muscle spindle. He concluded, therefore, that the spindle is innervated by fibers derived from the posterior root ganglion, and that it is a specialized sensory end-organ well adapted to the mechanical stimulations responsible for its reactions.

17. Dart, R. A.: Some Notes on the Double Innervation of Mesodermal Muscle, J. Comp. Neurol. **36**:441 (April) 1924.

The spindle has also been regarded as an organ designed for protection of the nerve terminals during contraction of the skeletal muscles and as a highly differentiated lymphatic body.

Furthermore, it is evident from the peculiarities of the intrafusal muscle, that the spindle possesses an unusual degree of intrinsic vitality. Sherrington excised a portion of the sciatic nerve in the cat and sutured the proximal end upward. No reunion of the nerve occurred, and the muscles were completely paralyzed. One hundred and fifty days after the operation, it was found that the extrafusal fibers in the gastrocnemius and plantar muscles were altered almost beyond recognition, and that all nerve fibers to the muscle spindles had completely disappeared; but "the intrafusal muscle fibers were not obviously altered." Every spindle examined remained thus unchanged. Sherrington, accordingly, expressed the opinion that in regard to their nutrition, the intrafusal muscle fibers appear to be independent of both the afferent and the efferent nerves to the muscle. Batten, likewise, found no degeneration of the muscle bundle three months after he had cut the sciatic nerve in the cat. Nevertheless, he was of the opinion that if a longer period were allowed for degeneration the intrafusal muscle would exhibit atrophic changes. This belief is contradicted, however, by Horsley, who discovered no abnormality of the intrafusal muscle one year after the sciatic nerve had been excised.

Thus, it is evident that the spindle is innervated from the posterior root ganglion, that there is no conclusive evidence of its direct innervation from the lower motor neuron or from the autonomic nervous system, and that the intrafusal muscle possesses a degree of nutritional independence not exhibited by the general striated musculature. The specific function of the afferent fibers from the spindle is not fully determined, but current opinion concerning the probable nature of the nerve impulse is not essentially different from that expressed by Barker,[18] in 1901: "The whole make-up of the muscle spindle or neuromuscular bundle impresses one as a structure especially adapted as a sense organ to give information concerning various states of tension in the muscle. Contraction of the muscles in which they are situated must necessarily lead to alterations in the pressure of the lymph inside them, and I am inclined to agree with those observers who assign to them an important function in connection with muscular sense."

THE MUSCLE SPINDLE IN VARIOUS PATHOLOGIC CONDITIONS

To this experimental evidence as to the nature and function of the spindle, may be added the knowledge derived from its study in a number of neuromuscular affections.

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18. Barker, L. F.: The Nervous System and Its Constituent Neurones, New York, D. Appleton & Co., 1901, p. 418.

Disease of the Lower Motor Neuron.—As early as 1876, Eisenlohr described what were apparently normal spindles in the much atrophied muscle of poliomyelitis. He was, however, unfamiliar with their meaning and regarded them as pathologic formations. Sometime later, Blocq and Marinesco, studying post mortem embedded material from a case of poliomyelitis found the spindle entirely normal and reported the observation as evidence of the integrity of the spindle in the otherwise completely degenerated muscle of this disease. In specimens of muscle obtained from six cases of poliomyelitis, Batten examined the spindle by the Sihler method and in microscopic sections stained by various methods. All of his material was obtained post mortem and five of the specimens were from old museum preparations which had been preserved in alcohol for many months. The sixth specimen was secured at necropsy. In each instance the paralysis had been of long standing, and all of the specimens showed great atrophy of the muscular tissue with occasional complete fatty disintegration. Spindles were found in the material from all of the cases except one, and all of them were entirely normal. The intrafusal muscle, and the nerve fibers and their terminals were well preserved.

Preservation of the spindle in the atrophied muscle of progressive central muscular atrophy has been noted by Batten, Pilliet,[19] Blocq, and Laura Forster. Both the muscular and the nervous tissue of the spindle were unaltered. Laura Forster has also found the spindles normal in a case of bulbar palsy.

Disease of the Corticospinal Tract.—Although the hypertonia of upper segment disease differs materially from that observed in the parkinsonian syndrome, it is significant that in the former type of rigidity with contracture Pilliet found the spindle unchanged. The specimen of muscle was secured at necropsy from one of the flexors of the thigh in a subject who had suffered from amyotrophic lateral sclerosis.

Disease of the Afferent System.—Since the nerve fibers from the spindle are conveyed to the central nervous system through the posterior spinal roots, it is presumable that the tabetic lesion might be productive of changes in the neuromuscular bundle; but only when the syphilitic process has invaded the ganglion cells or their peripheral axons. Batten has examined the spindles, both in teased and in sectioned material, from three cases of tabes dorsalis. In two of the cases all of the spindles were normal, while the specimens in the third case exhibited minor changes which he described in uncertain and contradictory words. In the body of his paper he states that the teased specimens showed no disease of either the nerve or the muscle, but the microscopic sections,

19. Pilliet, M.: Note sur des corps neuro-musculaires à enveloppe semblable à celle des corpuscules de Pacini, Soc. de Biol. Paris **42**:313, 1890.

stained by the Marchi method, exhibited fatty degeneration of the intrafusal muscle in the equatorial region. The nerve fibers in this region were of normal appearance, however, when stained by the Pal method. It is, therefore, difficult to discover the evidence on which Batten makes the concluding statement that "certain changes take place in the termination of the nerves, the general structure of the spindle remaining normal." He made no examination of the nerve roots, the ganglia or the nerve trunks. Schiefferdecker, however, refers to a study of the spindle in tabes by Langhans, who found all of the specimens normal in every particular.

Disease of the Spinal Cord.—In only two instances has the spindle been examined in focal or disseminated disease of the spinal cord. Babinski studied the muscle from a case of chronic diffuse myelitis, and Laura Forster obtained a specimen from a patient who died one year after the development of a complete transverse myelitis; but neither of these cases exhibited any change in the neuromuscular bundle.

Disease of the Peripheral Nerves.—The changes observed in the spindle following experimental excision of a peripheral nerve suggest that the spindle might also suffer in severe neuritis of long standing or in ununited nerve injuries; and, yet, examination of the muscle in these conditions has not revealed an invariable reaction in the neuromuscular bundle. Batten found the spindle unchanged in a case of mild neuritis, but he regarded the observation as untrustworthy, since the surrounding musculature was only slightly altered. He refers, however, to the studies of Eichhorst and of Siemerling, both of whom found the spindles normal in long-standing alcoholic neuritis; but Gudden, it is stated, noted slight degeneration of the intrafusal muscle and defective staining of the spindle nerves in this disease. Nevertheless, Pilliet, and Blocq and Marinesco found no change in the spindle in polyneuritic muscular atrophy.

That the spindle suffers a degenerative change after complete and ununited injury of the peripheral nerve is evident also in the studies of Batten. His specimens were obtained from a case in which motor and sensory paralysis of the arm had persisted for twelve months. All muscles of the arm were greatly atrophied, and sections of this tissue revealed small, contracted spindles which did not stand out in contrast to the surrounding atrophic muscle. The intrafusal muscle fibers were degenerated, and the nerve to the spindle stained faintly; these changes were also apparent in the teased specimens stained by the Sihler method. He concluded that in injury to the peripheral nerves the spindle undergoes atrophic degeneration, but at a much later date than the surrounding muscle, which in this instance was almost totally destroyed.

Toxic and Other Diseases.—Batten refers to Siermerling's study of the spindle in acute phosphorus poisoning, in which he found the intrafusal muscle fibers filled with fat; and it is especially significant that Askanazay, according to Schiefferdecker,[20] has observed changes in the muscle bundle in exophthalmic goiter, but the intrafusal nerve fibers were said to be unaltered. Laura Forster has examined the spindle in conditions other than those acting directly on the nervous system and found it unchanged in nephritis, in acute miliary tuberculosis, in valvular heart disease and in general sarcomatosis.

Disease of the Muscular Tissue.—It is, however, in the myopathies that the spindle has been most extensively examined, since the muscle tissue in this disease has been the object of numerous investigations. Within the same year Spiller,[21] Grünbaum,[22] and Batten, respectively, found the spindle unchanged in muscular dystrophy, in pseudohypertrophic paralysis and in Leyden's form of myopathy. In Spiller's case the intramuscular nerves were also normal, although atrophy and degeneration of the surrounding muscle were said to be intense. Grünbaum obtained his material at necropsy, but did not examine the tissue until nearly two years later. He then found the extrafusal muscle largely replaced by fat, but "the muscle spindle was for the most part unaffected," and the intrafusal nerve fibers were invariably normal. No change was noted in the spindle of the myopathic muscle when studied by Westphal and Santesson, and Siemerling found its structure unchanged in a case of progressive neural muscular atrophy.

With few exceptions, examination of the spindle in the human being has been confined to the use of "fixed," embedded and sectioned material obtained at necropsy; and, although all of his tissue was secured post mortem, Batten was probably the first to study the teased spindle in pathologic conditions. A more promising method was adopted by Sala [23] when he studied the muscle removed by biopsy from seven cases of pseudohypertrophic dystrophy. Notwithstanding his elaborate technic, it appears that the greater part of his material was embedded and cut into microscopic sections. Although some of his illustrations suggest that he might have examined an occasional teased specimen, the specific

20. Schiefferdecker, P.: Beiträge zur Kentniss der Myotonia congenita, der Tetanie mit myotonischen Symptomen, der Paralysis agitans und einiger anderer Muskelkrankheiten, zur Kentniss der Aktivitäts-Hypertrophie und des normalen Muskelbaues, Deutsche Ztschr. f. Nervenh. **25**:1, 1904.

21. Spiller, W. G.: The Neuro-Muscular Bundles, J. Nerv. & Ment. Dis. **24**: 626, 1897.

22. Grünbaum, H. S.: Note on Muscle Spindles in Pseudohypertrophic Paralysis, Brain **20**:365, 1897.

23. Sala, G.: Die pseudohypertrophische Paralyse, Arch. f. Psychiat. **55**: 389, 1915.

technic is not recorded and he confesses that satisfactory specimens were never obtained in the gold or methylene blue preparations.

In none of his material did he find a single spindle which was entirely normal in all its parts. Nevertheless, the changes were confined almost entirely to the muscle bundle, which, in cross and longitudinal sections, exhibited the abnormalities characteristic of the surrounding musculature, although the intrafusal nerve was invariably normal in the early stages of the disease. However, when the atrophy of the muscles was intense the nerve fibers in the spindle apparently suffered also, but the spiral and platelike endings, which seemed always more resistant, were usually preserved even when the spindle was otherwise greatly altered. No evidence of disease was apparent in the intramuscular nerve trunks, and Sala, therefore, regarded the change in the intrafusal muscle as the primary lesion. Babés and Kalindero, according to Sala, have likewise noted, in a case of myopathy, disease of the muscle bundle without evident lesion in the spindle nerve, and similar observations are said to have been recorded by Furstner and by Finkelnburg.

It is apparent, therefore, that the spindle possesses a peculiar resistance to disease; that, ordinarily, it does not share in the general pathology of the nervous and muscular systems and that the changes which have been noted in its structure are concerned largely with the intrafusal muscle fibers. Neuritis, traumatic or experimental injury of the peripheral nerves, tabes dorsalis, phosphorus poisoning, exophthalmic goiter and the myopathies are the only conditions in which the spindle has exhibited pathologic changes. In many instances the lesion was slight and of questionable nature; with the exception of tabes and exophthalmic goiter, it was apparent only when the atrophy of the surrounding musculature was intense. It is especially noteworthy that primary disintegration of the neuromuscular bundle has not been recorded in any disease.

THE MUSCLE SPINDLE IN PARALYSIS AGITANS

Examination of the spindle in paralysis agitans is inseparably connected with the study of the striated musculature by those who sought to establish the myopathic nature of the disease. These investigations revealed inconstant changes characterized by variable degrees of simple or degenerative atrophy, moderate fibrosis and proliferation of sarcolemma nuclei in the skeletal muscles. Lesions of this type have been described by Camp [24] who studied the tissue from eight cases of paralysis agitans, in one of which the specimen was obtained by biopsy from the muscles of the forearm; but these observations are contradicted in the

24. Camp, C. D.: Pathology of Paralysis Agitans with a Report of Fourteen Cases with Necropsy, J. A. M. A. **48**:1230 (April 13) 1907.

studies of Burzio,[25] and Redlich,[26] both of whom found the muscle fibers only slightly, if at all, affected. The muscle of the parkinsonian patient has been obtained from the living subject in only two other instances, recorded by De Buck and De Moor [27] and by Sana Salaris;[28] but in neither monograph is any reference made to the neuromuscular bundle.

Thus, while the muscular tissue in paralysis agitans has been repeatedly examined, it seems that the spindle has not been an object of special study. Only three references were found in the literature pertaining to this aspect of the pathologic process, and in each instance the lesion in the spindle was characterized by changes in the intrafusal muscle alone.

So far as I am aware, Idelsohn [29] was the first to note the condition of the spindle in paralysis agitans. His material was obtained at necropsy from the muscles of the hand and arm in one case, and although the sections revealed slight changes in the muscular tissue the intramuscular nerves, the muscle bundle and the nerves to the spindle were unaltered. Schiefferdecker later included in his study of the myopathies one case of paralysis agitans, from which the specimens were likewise obtained at necropsy. Each of the three spindles discovered in his sections exhibited slight lacunar degeneration of the muscle bundle and moderate thickening of the capsule, but the intrafusal nerve fibers were normal with both myelin and axis-cylinder stains. He was of the opinion that the lesion in the muscle bundle was independent of the changes in the surrounding musculature and that the disease of the intrafusal muscle was primary and peculiar to paralysis agitans.

Finally, in Camp's study of the muscle, it is not clear to what extent he examined the spindle, how many were found in his specimens, or whether they were studied in postmortem material or in tissue removed at biopsy. He states that "the muscle spindles were hard to find in the sections from any of the cases, but in those examined the muscle fibers within them showed similar changes to those surrounding them. The nerve fiber within the spindle was normal and the intramuscular nerves showed no degeneration when stained with the Weigert haematoxylin stain."

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25. Burzio, F.: Alterazioni anatomiche ed istologiche del sistema nervoso nel morbo di Parkinson, Ann. di freniat. **12**:151, 1902.

26. Redlich, E.: Beitrag zur Kentniss der pathologischen Anatomie der Paralysis Agitans und deren Beziehungen zu gewissen Nervenkrankheiten des Greisenalters, Jahrb. f. Psychiat. **12**:384, 1893-1894.

27. De Buck and De Moor: Malade atteint de maladie de Parkinson, Bull. Soc. de méd. de Gand. **66**:113, 1898-1899.

28. Salaris, S. G.: Note di istologia patologica (per biopsia) sulla fibra muscolare striata nel morbo di Parkinson, Riv. di patol. nerv. **10**:383, 1905.

29. Idelsohn, M.: Lésions musculaires dans la maladie de Parkinson, Rev. neurol. **12**:394, 1904.

It is apparent, therefore, that the neuromuscular bundle has not been extensively studied in paralysis agitans, and I found no record of its examination in the fresh, teased material obtained either during life or at necropsy. These circumstances, together with the statement by Sherrington that "the acidity developed in muscles post mortem quite rapidly seems to alter the myelinate fibers in the nerve twigs," led me to believe that more precise knowledge of the spindle might be derived from examination of the teased specimen obtained from tissue in which changes of the foregoing character could be largely excluded.

SOURCES OF THE MATERIAL, CLINICAL ABSTRACTS AND TECHNICAL METHODS

A specimen of muscle was obtained from the living subject in each instance and immediately transferred into freshly prepared solutions of "fixing" and staining reagents most suitable for the study of muscle and nerve. Material was thus secured from fourteen cases of genuine paralysis agitans, from one case of the postencephalitic parkinsonian syndrome and from a healthy person. From Mr. C. H. Miller of the Carnegie Institute, I obtained a mounted normal specimen taken at necropsy from the intercostal muscle of a child, and I am further indebted to him for the preparation of material from several of my earlier cases.

Eight of the cases of paralysis agitans were derived from my private practice; the remaining six were seen in the Johns Hopkins Hospital Dispensary, where permission was obtained to transfer them to the Church Home and Infirmary for further study. Of the fourteen patients, eleven were men and three women. The ages varied from 41 to 70; ten were in the fifth decade; three in the fourth, and one in the seventh. In no case was the disease preceded by a constitutional or infectious disorder. None of the patients admitted since 1918 gave an account of any illness suggestive of epidemic encephalitis, and although a negative history does not preclude the possibility of a remote evanescent attack of the disease, in no instance were the development or progress of the parkinsonian symptoms comparable to those commonly observed in the postencephalitic affection.

In five cases the symptoms were initiated by tremor confined to the thumb, the middle finger or the hand for variable periods, during which the disease made no further progress. In four instances tremor was the outstanding feature, in two it was slight and only occasionally present, while in the remaining cases it was of moderate intensity. Rigidity dominated the clinical picture in five of the patients; in others, tremor and rigidity were equally conspicuous. The duration of the symptoms varied from one to nine years, and both unilateral and bilateral types

of the affection were represented. Opportunity was thus afforded to examine the muscle in the early and in the late course of the disease, in the tremulous and in the rigid phase of the disorder and in the classic syndrome with its admixture of tremor and rigidity. I shall review here only the essential clinical features of four cases. The first specimen was obtained, July 3, 1916, from the extensor muscles of the left forearm. No spindles were found in this material. In the meantime, I learned of their common occurrence in the small muscles of the hand, and all specimens from subsequent cases were obtained from the abductor pollicis brevis of the more affected hand.

REPORT OF CASES

CASE 1.—Oct. 3, 1916. W. Z. W., a man, aged 51, suffered from "severe trembling," which made its appearance five years previously and was at first confined to the thumb of the left hand, but within twelve months had involved successively all fingers, the hand and forearm on this side. During the second year of his illness, stiffness and slight tremor developed in the left leg, and within the following two years the fingers of the right hand, the entire upper extremity and, to a lesser degree, the lower extremity on this side were consecutively and similarly affected. Stiffness of the muscles of the neck, defective speech and the characteristic parkinsonian facies and attitude were then gradually acquired. Loss of automatic and associated movements was apparent, but muscular rigidity was not a prominent symptom.

Examination revealed no abnormalities other than those commonly observed in paralysis agitans. The tremor was incessant; and although the muscles were usually quiet when the patient slept, he stated that occasionally the tremor awakened him from a sound sleep. At times the trembling was so great as to shake the ordinary hospital bed if the patient lay on it while he was awake, but during voluntary movement the tremor was scarcely visible. Physical examination of the thorax and abdomen by Dr. L. Limbaugh of the Church Home and Infirmary, and laboratory studies of the blood, the gastric juice, the urine and the spinal fluid failed to show any abnormality. The specimen of tissue, removed by Dr. E. H. Teeter, Oct. 16, 1916, from the left thenar muscles, contained four spindles.

CASE 2.—May 20, 1918. J. D. C., a man, aged 70, had always enjoyed excellent health until November, 1916, when he noticed trembling of the right hand. Within a few months the tremor involved the entire right upper extremity and, to a slight degree, the left hand and forearm. He later complained of stiffness of the neck and stated that his friends then noticed a change in the expression of his face. He exhibited the typical parkinsonian mask and a tremor during rest affecting both upper extremities, but more especially the right. Because of the rigidity, voluntary movement of the arms was slowly executed, and inhibition of automatic and associated acts was particularly noticeable in the right upper extremity. The tremor was greatly diminished or abolished on voluntary movement. With the exception of general arteriosclerosis and vascular hypertension, physical examination revealed a remarkably well-preserved man. The laboratory studies of blood, urine, gastric juice and spinal fluid gave negative findings. Three spindles were found in the specimen of muscle obtained from the right thenar eminence by Dr. Teeter, May 24, 1918.

Case 3.—Dec. 5, 1923. K. K., a woman, aged 59, complained of "stiffness and shaking," first noticed two years previously, and at first confined to the right hand. Within a few weeks the left hand was similarly affected. Six months after the onset, stiffness of both legs was prominent, and rigidity of the arms had almost entirely replaced the tremor. At the time of examination the masklike facies, the retarded speech and the great rigidity of most of the musculature were outstanding features, though the date of their onset is unknown. Tremor of the hands was scarcely perceptible, and all movements were slow and deliberate, so the patient experienced much difficulty in arising from the sitting posture. Associated and automatic movements were completely abolished.

Physical and laboratory study by Dr. W. L. Clark of the Church Home and Infirmary revealed no abnormality, and my examination of the nervous system disclosed no evidence of disease other than the characteristic parkinsonian symptoms.

Dec. 12, 1923, Dr. Panettiere secured a specimen from the right thenar muscle. Twenty spindles were found in this material, sixteen in the teased tissue and four in the embedded and sectioned material. Of the twenty spindles, only four retained any resemblance to the normal.

Case 4.—Dec. 10, 1923. G. D., a man, aged 63, had difficulty in using his hands and feet, which he attributed to stiffness that had been present during the last year. In the preceding seventeen years his health had been good. The disease made its appearance after a very hard day's work, when he noticed that his hands began to tremble and he "seemed to go all to pieces." Within a few weeks stiffness developed in all the extremities accompanied by slight soreness in the muscles of the legs. With the progress of the rigidity, the tremor became less apparent and was often abolished for prolonged periods, but he could walk for only a short distance and then "had to shuffle along," because his legs "felt tied up." Recently, he had been unable to rise from a chair without assistance, and his body had become "bent forward."

Physical and laboratory studies by Dr. Clark showed no abnormality in the blood or secretions, and the neurologic examination revealed the features characteristic of the rigid type of paralysis agitans. At times the rigidity was so great that the patient could scarcely walk unless his inertia was suddenly overcome by a vigorous push, but tremor was not apparent unless he was unduly excited. Speech was slow and hesitating.

A portion of muscle was removed from the right thenar eminence by Dr. Panettiere, Dec. 12, 1923. Fourteen spindles were found in the teased preparation and four in the embedded material. Every spindle was greatly altered, and some were scarcely recognizable.

Normal Case.—A specimen of normal tissue was removed by Dr. Panettiere, Nov. 2, 1923, from the right thenar muscles of T. L., a man, aged 41, who three years previously had suffered a slight stroke with resulting left hemiparesis, from which he had largely recovered. He was at the time in good general health and was actively employed. Neither extremity on the right side had ever exhibited any nervous or muscular affection. The physical and neurologic examinations revealed no abnormalities other than those consequent on the previous vascular cerebral accident.

TECHNIC

For the operative procedure followed in securing the specimens of muscle, I am indebted to Dr. H. L. Homer of Baltimore, and to Drs. E. H. Tetter.

Hiram Fried, R. G. Coblentz and C. Panettiere, each of whom was, at one time, resident surgeon at the Church Home and Infirmary. Under local procaine anesthesia a skin incision 4.5 cm. long was made over the belly of the abductor pollicis brevis parallel to the course of its fibers. The muscle was exposed by blunt dissection, and silk ligatures were placed about 0.5 cm. apart at its proximal and distal attachments. When the ligatures were tied they enclosed a fasciculus of muscle almost 1 cm. in thickness. The tissue was then cut between the two ligatures at each end of the muscle, and the specimen was lifted from the wound by the two proximal threads. By this means a portion of muscle was secured which measured approximately 3.5 cm. in length and 1 cm. in thickness, and which was subjected to the least possible injury during removal.

The specimen from each case was immediately cut transversely into a number of nearly equal portions which were placed respectively in the following solutions: Sihler's solution 1; lemon juice (Ranvier method); 1 per cent. gold chloride (Cohnheim method); Zenker's fluid; alcohol, 70 per cent.; saturated solution of mercuric chloride; and solution of formaldehyde 4 per cent. Since alcohol and mercuric chloride fixation possessed no particular advantage over the Zenker method, they were later discarded. The Zenker and the formaldehyde treated material were so embedded in celloidin as to procure both cross and longitudinal sections.

Most of the material was cut at 6 microns, and every tenth section was examined under the dissecting microscope for spindles. When one was found, the subsequent sections were similarly examined until the entire spindle had been sectioned. With the exception of one instance, in which the tissue was embedded in paraffin, no effort was made to keep the sections in series, but every section containing a portion of a spindle was preserved; and all material of this character from each case was later stained and mounted. The following staining reagents were used: Ehrlich's acid hematoxylin and eosin, hematoxylin and acid fuchsin, hematoxylin and van Gieson stain, phosphotungstic acid-hematoxylin, the Alzheimer-Mann stain, Weigert's myelin stain and the Bielschowsky silver method. Material treated by the Sihler, the Ranvier and the Cohnheim methods was studied entirely in teased dissections; but some of the spindles isolated from these preparations were subsequently stained with Scharlach R, and by the Marchi method.

When a successful gold impregnation is obtained by either the Cohnheim or the Ranvier method the result is highly satisfactory; but it is well known that the gold technic is not always reliable, and that, occasionally, it fails to impregnate normal tissue. This is not true, however, of the Sihler method, and I have never failed to obtain by this precedure a successful preparation. Since the method is not widely used by neuropathologists, it may be well to review, briefly, its essential steps as recorded by Batten. Two solutions are employed:

Sihler's Solution no. 1

Glacial acetic acid	1 volume
Glycerin	1 volume
Chloral hydrate 1 per cent. aqueous solution	6 volumes

Sihler's Solution no. 2

Ehrlich's acid hematoxylin	1 volume
Glycerin	1 volume
Chloral hydrate 1 per cent. aqueous solution	6 volumes

Either fresh or early necropsy material may be used. Portions of partly teased muscle, not larger than 2 by 1 cm., are placed in solution 1 for eighteen hours, when they are again teased in pure glycerin and transferred to solution 2 for from three to ten days. During this period small fragments of the tissue should be examined at intervals. Since both the muscle and the nerve are deeply stained by the hematoxylin, differentiation is necessary, and this is accomplished by replacing the material in solution 1 for from fifteen to thirty minutes. The muscular tissue is thus decolorized until it becomes pale blue, or light brown if left in the solution for the longer period; but the nerve fibers do not lose their coloring even after prolonged differentiation. I have allowed specimens of muscle to remain in the differentiating solution over night, and yet the normal nerve retained its deep blue color, while the diseased nerve was no more decolorized than after the shorter period of differentiation.

ISOLATION OF THE SPINDLE; ITS APPEARANCE IN THE NORMAL HUMAN MUSCLE

Before describing the changes in the diseased spindle, it will be instructive to examine the normal specimen as it appears in healthy muscle obtained from the living man, and subjected to the same technic as that adopted for the study of the pathologic material.

The specimen from the normal muscle was slightly smaller than those usually obtained from the cases of paralysis agitans and the number of spindles in this material was small. No spindles were found in the tissue prepared by the Ranvier method, but three well-stained specimens were isolated from the Sihler material and one was discovered in the Cohnheim material. These vary in length from 5 to 8 mm., and the equatorial diameter measures from 0.4 to 0.6 mm. In the embedded tissue, six spindles were apparent in the transverse and longitudinal sections; one is a specimen of the compound type. Thus with the seven specimens contained in the microscopic sections and the four derived from the teased tissue, a total of eleven spindles was found in a comparatively small piece of muscle.

Unless one has had experience in teasing the spindle from the surrounding muscle and in recognizing it with the unaided eye, or under low magnification, it might easily escape detection, since the illustrations by which it is represented in available literature have little resemblance to the spindle as it appears in the material.

A small piece of muscle, not more than 2 or 3 mm. in thickness, is teased in glycerin under the dissecting microscope; and discovery of the spindle will be facilitated if the teasing follows the course of one of the intramusclar nerve trunks. At first, the nerve and muscle are separated with comparative ease; but, as the smaller nerve fibers are approached the connective tissue usually becomes more abundant and retards the teasing. The dissection should then proceed with care, for it is generally in this mass of connective tissue that the spindle is found. In fact, the increased difficulty in isolating the nerve fibers may be due to the unsuspected presence of the spindle capsule, and if the tissue is handled roughly the nerve may be torn from the capsule, or the entire spindle may be destroyed. When this stage of the dissection is reached, if the spindle is not distinctly visible the fragment of muscle should be placed between two slides. These are firmly pressed together and the spindle, if present, can then be seen with the naked eye as a dark fusiform, elongated or crescentic body.

It is not always possible, by this means, to distinguish the spindle from the surrounding muscle, especially in the Sihler material which has not been thoroughly differentiated. The compressed tissue should then be examined

under low magnification with the compound microscope, and if a spindle is detected, the material should be transferred to glycerin and the dissection completed under the microscope. The nerve should be cut with scissors rather than torn apart with dissecting needles. The completely isolated spindle is then mounted in glycerin; its appearance under low magnification is shown in Figure 1. The individual fibers of the muscle bundle are indistinguishable except in the polar regions, and the intrafusal nerve can be traced for only a short distance after it pierces the capsule. A blood vessel and a nerve trunk are shown entering the left polar region of the spindle.

Under higher magnification, Figure 2, the deeply staining nerve fibers are seen coursing along and encircling the intrafusal muscle. Only the central portion of the specimen is represented in the illustration, and because of the frequent loss of striations and accumulation of nuclear material in this region, the individual muscle fibers are still indistinguishable. Both the nerve and the muscle are deeply stained, and the nerve fibers, although of a mauve color in this magnification, are dark blue or almost black when isolated and examined more closely in the well-stained specimen.

The detailed structure of the intrafusal muscle with its striations, nuclei and nerve terminals is readily discerned in the illustration, Figure 3, of the greatly enlarged left polar extremity of this spindle. Here, it is apparent that in the normal specimen the nerve fibers proceed to the end of the spindle where, although not so numerous, they stain with an intensity of coloring equal to that observed in the equatorial region. The muscle fibers, of which there are three, vary in size and contour, and their striations are often obscured by one of the various types of nerve-endings. That the nerve terminals are readily demonstrated at both extremities of the normal spindle is especially noteworthy; this feature is clearly visible on the large central muscle fiber and on the lowermost fiber where the nerve-endings have largely obscured the muscle striations.

The internal anatomy of the spindle can be satisfactorily studied only in microscopic sections (Fig. 4). Although embedded in the muscle substance, the spindle is separated from it by a connective tissue capsule. The size of the spindle, the thickness of the capsule and compactness of its lamellae, the occasional capsular nuclei and the absence of material in the interlamellar spaces are to be noted. Excentrically placed within the capsule, are three intrafusal muscle fibers surrounded by the axial sheath which separates them from the periaxial space; but the delicate fibrillar network, which sometimes extends across this space, is not apparent in the photomicrograph. Inside the capsule, but below and to the right of the intrafusal muscle, is a mass of tissue, the details of which are not distinguishable in this magnification. This is composed of connective tissue, nerve fibers and blood vessels.

The difference in size and structure of the extrafusal and the intrafusal muscle fibers is apparent. The latter exhibit a formation which has occasionally been regarded as evidence of degeneration; but this feature of the muscle is more clearly demonstrated in the higher magnification of this section, illustrated in Figure 5. Here, two of the fibers seem to be entirely replaced by a vesicular nuclear material, and one of them contains a central core of deeply staining muscle encircled by a number of vesicular nuclei. This appearance is due to the equatorial nuclear mass which surrounds or is embedded in the intrafusal muscle. The axial sheath with its nuclei, the structure of the capsule and the relative clearness of the periaxial and interlamellar spaces are also here more readily distinguished.

Those who have studied the spindle in various diseases assert that not only may a lesion be found in the intrafusal muscle, but that sometimes the entire spindle may be altered in size, shape and general configuration. With few exceptions, however, and never in the case of paralysis agitans, have these assertions been supported by actual measurement of the spindle or its component parts. Hence, in order to verify these statements and to provide an approximate standard of comparison for the spindle in paralysis agitans, I have measured, with the stage micrometer, sections from the pole, from the equator and from various intermediate levels in eight of the spindles obtained from the healthy muscle.

The diameter of the spindle was determined in each instance by measuring the widest portion of the section from the outermost lamella of the capsule; this measurement included, therefore, the width of the capsule. The greatest capsular thickness was then recorded separately, and the width of the periaxial space was determined by measuring the greatest distance from the axial sheath to the innermost lamella of the capsule. In many sections, the number and size of the intrafusal muscle fibers were determined, and the average diameter of these was tabulated with similar measurements of the extrafusal musculature. The size of the intrafusal nerve fibers was also determined in many sections and, occasionally, the diameter of the blood vessels within the axial sheath was recorded.

In all instances the micrometer readings have been converted into millimeter equivalents, and are recorded as such in the following summary: The total diameter of the spindle varied from 0.018 mm. in the polar region to 0.178 mm. at the equator, where it had an average diameter of 0.132 mm. At the extremities of the spindle, the capsule measured 0.0066 mm. in thickness but it occasionally attained a width of 0.042 mm. in the equatorial region where it was usually not more than 0.025 mm. The periaxial space, likewise, had its greatest dimension at the equator, where it measured as much as 0.036 mm., although an average width of 0.030 mm. was obtained for the eight spindles. The wall of the blood vessels in the axial sheath had an average thickness of 0.0024 mm. The diameter of the larger intrafusal nerve fibers varied from 0.0012 to 0.0014 mm. Each spindle contained from three to six muscle fibers which measured from 0.0028 to 0.028 mm. in diameter.

In the Bielschowsky preparation of a longitudinal section, Figure 6, several healthy axis-cylinders measuring 0.0014 mm. in diameter are visible within the axial sheath and on the muscle tissue. The transverse striations of the intrafusal muscle and the intramuscular nuclei are also easily recognized. Because of the frequent contraction of the tissue during the embedding process, it is difficult to secure a section through the entire longitudinal axis of the spindle, and, unfortunately, all specimens from the tissue, which was embedded for longitudinal sectioning, were cut in oblique fashion (Fig. 6). It was impossible, therefore, to obtain reliable measurements of the longitudinal dimensions of these spindles. Nothwithstanding this source of inaccuracy the largest section of the spindle measured 0.264 mm. in its greatest dimension; the periaxial space attained a width of 0.075 mm., and the diameter of the intrafusal muscle fibers was not greater than 0.018 of a millimeter. The largest axis-cylinder measured 0.0016 in thickness.

Although the dimensions of the spindle are slightly smaller in the child than in the adult, there is no material difference in structure. I have therefore included an illustration, Figure 7, of a normal specimen from the intercostal muscle of a child, since it furnishes a more satisfactory picture of the spindle

in longitudinal section. A large part of the equatorial region is included in the section, in the center of which the muscle bundle with its nuclei and axial sheath are apparent. Immediately above the right extremity of the intrafusal muscle, but separated from it by a slight space, are a number of axis-cylinders within the axial sheath. Notwithstanding the obvious enlargement of the drawing, the muscle striations and the individual fibers of the muscle bundle were not apparent under the actual magnification from which the sketch was made; and, furthermore, the division of the muscle in the equatorial region very often obscures their individuality. The appearance of the periaxial space, the delicacy of the tissue forming the capsule and the axial sheath and the relatively small amount of very fine fibrous strands extending here and there across the periaxial space are to be noted especially; and if the normal spindle is compared with the diseased specimen, a striking change is apparent in its dimensions and structure.

THE PATHOLOGY OF THE SPINDLE IN PARALYSIS AGITANS

Before describing the changes in the internal structure of the neuromuscular bundle, it will be instructive to refer to some of the general characteristics of the pathologic material.

With the exception of the specimen of muscle secured from the forearm of the first patient, spindles were obtained in every instance; but the number of spindles discovered in the tissue from my earlier cases was never so great as that derived from my later studies when I had learned to recognize more readily the diseased spindle in the teased material. Thus, in a total of sixty-seven spindles from the thirteen cases, thirty-eight were found in the material from the last two patients; although not all of the spindles in any one case were affected, specimens exhibiting the characteristic lesion were found in each instance. Moreover, the spindles were affected in variable degree, so that it was not uncommon to obtain, from a single portion of muscle, a variety of specimens illustrating different stages in the development of the pathologic process.

There was evidence, also, that the number of diseased spindles and the intensity of the morbid change depended less on the duration of the illness than on the dominant clinical feature of the disorder. Thus, it was occasionally observed that a greater number of spindles were affected, and that the lesion was of a more destructive character in cases in which the disease was of only one year's duration, than in those in which it had persisted as long as five years. Regardless of the duration of the illness, it seemed also that when tremor was the outstanding symptom, the lesion in the spindle was never advanced to as great a degree as when tremor had been replaced by great rigidity, or when rigidity was the dominating symptom from the outset; and although an occasional normal spindle was found in the latter instance, the lesion was always wide spread and destructive. Hence, in describing the early changes in the spindle, reference is made not to the duration or character of the clinical symptoms, but to the first evidences of a lesion in the neuromuscular bundle.

In the beginning, the entire spindle is swollen from what appears to be edematous distention of the periaxial and interlamellar spaces; this enlargement of the spindle is distinguishable both in the teased and in the sectioned material. Examination of the section, reproduced in Figure 8, reveals also an accompanying proliferation of the connective tissue elements in the capsule and axial sheath. The overgrowth of connective tissue has invaded the interlamellar and periaxial spaces, and is especially conspicuous in the latter just above and below the intrafusal muscle. Likewise, there is thickening of the

delicate fibrillar tissue in the periaxial space, which also contains an accumulation of granular material similar to that occasionally found in small amounts in the normal spindle.

The intrafusal muscle fibers are only slightly altered. They are of normal size, but take the stain unevenly and are irregular in outline. The two more centrally placed fibers exhibit beginning lacunar degeneration; but the enlargement of the spindle and hyperplasia of the connective tissue are the distinctive features. Although the cross sections of several normal axis-cylinders are visible in the endomysium below and to the right of this spindle, there is no evidence of nerve tissue in the capsule or axial sheath. The extrafusal musculature is of healthy appearance.

In some specimens, the distention of the spindle is so great that the periaxial space alone attains a width almost equal to the total diameter of the normal spindle. The cross section of the spindle from Case 2, illustrated in Figure 9, was photographed at the same magnification as that of the normal spindle shown in Figure 4. The enormous distention of the periaxial space has apparently compressed the capsular lamellae and prevented corresponding distention of the interlamellar spaces, so that the capsule is not relatively increased in thickness. The periaxial space contains a large amount of granular material and a detached capsular lamella. The hyperplasia of the connective tissue is not easily demonstrated in a photograph at this magnification, but the sclerosis of the axial sheath and its blood vessels, and degeneration of the muscle bundle, are apparent. A partly degenerated portion of the equatorial nerve trunk is distinguishable, within the capsule, in the constricted portion of the spindle below and to the extreme left of the muscle bundle, but the axial sheath contains no nerve fibers. Although the greater part of the extrafusal musculature is of normal appearance, there are in the immediate vicinity of the spindle a few fibers in a state of simple atrophy.

A higher magnification of the central portion of this spindle, Figure 10, portrays more distinctly these early structural changes. Here, the sclerosis of the axial sheath and its blood vessels, the proliferation of fibrous tissue in the periaxial space and the changes in the muscle bundle are unmistakable. The thickening of the vascular sheath is, in all probability, only part of the general arteriosclerosis apparent in the patient from whom this specimen was obtained in his seventieth year. This picture may be compared with that of the normal spindle, Figure 5, photographed at the same magnification. Although the intrafusal muscle fibers in the pathologic specimen have retained their normal size—the largest is not more than 0.017 mm in diameter—they are obviously in a condition of degenerative atrophy. The central nuclear material is not apparent, and the muscle is represented by a mere ring of sarcoplasm enclosing a central mass of degenerated tissue, or by a crenated fragment of muscle surrounded by a clear space of variable width; but these central and marginal zones, unlike those in the normal specimen, are devoid of nuclei. In none of the sections from this spindle was there evidence of nerve fibers or nuclear material in the muscle bundle.

Nevertheless, these changes in the intrafusal muscle are not distinctive; they are often not apparent in a spindle which is otherwise greatly altered; so long as my study was confined to examination of the embedded and sectioned tissue, no convincing evidence of a specific lesion was discovered in the neuromuscular bundle. The essential nature of the pathologic process is disclosed, however, in a study of the teased specimens, in which it is evident that the changes in the muscle bundle are late manifestations, consequent on degeneration of the intrafusal nerve.

Apparently, the degeneration of the nerve is preceded by swelling of the spindle, and this early stage of the lesion is represented in the photomicrograph, Figure 11, of a teased specimen from Case 3. The equatorial nerve trunk is well stained and, with the aid of the microscope, its nerve fibers can be traced to both extremities of the spindle. An accompanying blood vessel crosses the spindle outside the capsule and to the left of the equator. The central nuclear material and the intricate nerve plexus in this region obscure the definition of the intrafusal muscle which, on higher magnification, presents a normal appearance throughout. Although the spindle has a length of only 5 mm., its equatorial width is 0.714 mm., whereas in the normal teased specimen of twice this length, 0.5 mm. is the greatest recorded diameter.

It is particularly significant that in a compound spindle, obtained also from this patient, one of the specimens has retained its normal structure in all details, while the other exhibits the early changes of the pathologic process. Thus, in the double spindle, Figure 12, the swelling of the equatorial region in the upper specimen is readily appreciated when contrasted with the same portion of the normal spindle, below and to the right. During the process of teasing it was not at first recognized that the two spindles were conjoined, and in the attempt to isolate them, the upper spindle was torn from its attachment in the right polar region where the two capsules were united. The critical observer may assert, therefore, that the swelling is only apparent and the result of trauma, or that the upper spindle is of greater length and accordingly larger in all dimensions. Since the capsular tissue ordinarily contracts after being stretched or injured, and the difference in the length of the two spindles is not great, it is my opinion that the equatorial swelling in the upper specimen is not the result of injury to the capsule.

The muscle bundle is normal in each specimen, but the intrafusal nerve fibers in the two spindles are not impregnated to the same extent. In the lower specimen they are well stained and can be traced to both extremities of the spindle, whereas in the adjoining spindle, although well impregnated at the equator and for a short distance in each polar direction, they cannot be followed to the end of the muscle bundle as in the normal specimen.

In my study, spindles similar to that illustrated in Figure 13 were discovered, not only as isolated specimens, but in immediate proximity to a spindle with perfectly impregnated nerve fibers. Occasionally, specimens of the double spindle were observed, in which one spindle presented a normally stained nerve throughout, while the other failed to show evidence of nerve fibers within the capsule. By the Ranvier method, impregnation of the nerve ceased just beyond the capsular margin. All spindles of this type were preserved unmounted, and several of them were subjected to various technical manipulations in an effort to secure further impregnation of the nerve; these procedures were unsuccessful, and the failure of impregnation was, accordingly, attributed to the uncertainty of the gold chloride technic.

Since the Sihler method was found to be more reliable for the demonstration of the nerve fibers, some spindles in which the nerve was unsuccessfully impregnated in the gold solution were later stained according to this method. One of the spindles so treated is shown in Figure 14. When the specimen was teased from the Ranvier material, it presented a diversity of coloring resembling that illustrated in Figure 13, but the only evidence of nerve tissue was represented by the four isolated fragments still apparent in the composite drawing of the spindle after it had been counterstained. The spindle was first sketched as it appeared when teased from the gold solution, and subsequently resketched and colored according to its appearance after removal from the Sihler solution.

In the illustration of this doubly stained spindle, the central nerve trunk, accompanied by a blood vessel, is apparent in the equatorial region. The first two centimeters of the distal portion of this nerve are more deeply colored than the proximal and intracapsular portions. Within the equatorial region, superimposed on the intrafusal muscle, is a fragmentary loop of deeply stained nerve tissue. To the left of the equator, in the lower portion of the spindle, there are two additional nerve fragments; these, also, are deeply stained and are superimposed on the muscle bundle where they are arranged parallel to the longitudinal axis of the spindle. No other evidence of nerve tissue was apparent in the spindle after the gold impregnation. The polar nerve trunk to the right of the equator was visible, but its intracapsular fiber, as well as the proximal and intracapsular portions of the equatorial nerve, were not then distinguishable. When the spindle was counterstained in the Sihler solution, that portion of the equatorial nerve between the darker distal segment and the central equatorial loop became apparent, and the fine nerve fiber from the polar innervation to the end of the spindle was also added to the picture. Notwithstanding this evidence of nerve disintegration, the intrafusal muscle retained its normal structure and appears as a dark elongated body extending throughout the length of the spindle. The swelling in the equatorial region is unmistakable, and the spindle there attains a width of 0.720 mm.

It is conceivable that the previous treatment of the tissue by the Ranvier method had so altered the nerve fibers as to interfere with their successful demonstration by the counterstaining method employed; therefore, I shall direct attention to another imperfectly stained specimen, Figure 15, to which this objection cannot be made. Although prepared according to the Sihler method alone, the entire spindle is faintly stained; defective staining of the nerve, as well as reduction in the amount of nerve tissue, is more readily appreciated when the drawing is compared with that of the normal spindle stained by the same method. The equatorial nerve trunk contains two vertically directed fibers which proceed toward the spindle. The larger fails to take the stain at the capsular margin and, although the smaller nerve fiber to the right enters the capsule, it is faintly visible for only a short distance in the central portion of the spindle. No intrafusal nerve fibers are apparent in either polar region, and the extracapsular nerve in the equatorial region, running parallel to the longitudinal axis of the spindle, does not seem to contribute branches to the muscle bundle. The striations, nuclei and contour of the intrafusal muscle are normal.

A number of spindles of this type were subsequently stained with osmic acid, but the preparations were not altogether satisfactory; it was difficult to determine whether or not the variable amount of black droplets distributed along the course of the intrafusal muscle and in the periaxial space were products of nerve degeneration. This technic failed to disclose evidence of nerve fibers within the spindle. It is possible that the Sihler solution may have so altered the myelin as to inhibit the action of the osmic acid, although the extrafusal fat in the surrounding connective tissue was well stained.

That the intrafusal nerve fibers undergo actual degeneration comparatively early in the development of the pathologic lesion, is unmistakably evident in the section illustrated in Figure 16. The drawing is that of a longitudinal section through the equatorial region of a spindle in which the swelling is so great that the specimen was quite apparent to the unaided eye in the sectioned material. The unusual size of the spindle is more readily perceived by com-

paring the illustration with that of the normal spindle represented in Figure 7. The diseased spindle is magnified 173 diameters, the normal spindle 852, but the projected drawings of the two specimens are of approximately the same dimensions.

The internal anatomy of the spindle is also greatly altered. Extensive proliferation of the connective tissue is a conspicuous feature; the axial sheath is much thickened; the intracapsular fibrillae are increased in number and size, and the periaxial space is largely filled by this overgrowth of fibrous tissue. The intrafusal nerves and blood vessels also take part in this widespread sclerosis which is characteristic of the spindle lesion when tremor is the predominant clinical feature.

Extending longitudinally through the center of the section is a single muscle fiber which takes the hematoxylin stain unevenly and is partly constricted by the surrounding scar tissue. Although its striations are still apparent, the right third of the fiber is devoid of sarcoplasm and contains mere shadows of the original equatorial nuclear material which has lost its staining property and is represented by a collection of circular bodies containing a variable amount of granular débris. Immediately above the left extremity of this elongated muscle fiber, situated in the axial sheath, are three fragments of faintly stained muscle tissue also in a process of degeneration; beneath its central portion is a larger and more deeply stained remnant of intrafusal muscle, reduced to a fibrillar structure without striations or nuclei. The extrafusal musculature is well preserved.

Directly above the right extremity of the degenerated muscle bundle, attached to the capsule of the spindle and occupying most of the periaxial space in this region, is an ovoid mass of fibrous tissue recognizable in the section as a completely degenerated and sclerosed bundle of intrafusal nerve fibers. Above and slightly to the left of this fibrosed area, but outside the spindle capsule, is a lighter circular area which contains the cross sections of several partly degenerated axons from the extracapsular portion of the spindle nerve.

In the central portion of the section, immediately above the junction of the right and middle thirds of the elongated intrafusal muscle and situated within the axial sheath, are the longitudinal sections of four axial nerve fibers in process of degeneration. This feature of the lesion is unmistakable in the higher magnification of this area illustrated in the insert beneath the spindle. The drawing includes the four nerve fibers and, as a further aid in orientation, a portion of the intrafusal muscle with its unstained sarcoplasm and degenerated nuclear material. Here, the change in the axis-cylinders is apparent. The axons are irregularly swollen and attain a width of 0.0075 mm., whereas the diameter of the axis-cylinder in the normal spindle is not greater than 0.0025 mm.; in the vertically directed fiber to the extreme left, the axis-cylinder is completely degenerated. Two additional axons, also exhibiting degenerative changes, are visible just above the three vertical fibers.

Degeneration of the intrafusal nerve is further demonstrated in a section from this spindle, Figure 17, stained by the Weigert myelin method. Here, the hyperplastic connective tissue has almost entirely replaced the normal contents of the spindle; the muscle bundle is disorganized, and there are few remaining evidences of the spindle nerve. Within the rectangular area near the right extremity of the section are several intracapsular nerve fibers but these are in a state of disintegration, and the changes in the myelin are portrayed in the enlarged sketch of this area inserted above the spindle. There is, however, almost total absence of nerve fibers in the periaxial fibrous tissue; many

of the deeply stained objects visible in this region are cross-sections of smaller blood vessels. In the center of the spindle, parallel to its longitudinal axis and near the left extremity of the section, are several fragments of the muscle bundle. Just above the equatorial end of this intrafusal muscle and within the periaxial tissue, there is a greatly sclerosed area containing a blood vessel with four myelinated fragments to its left and a completely degenerated fasciculus of nerve fibers to its right.

Thus far, the pathologic changes in the spindle are characterized by edema of the periaxial and capsular spaces, disintegration of the intrafusal nerve fibers, hyperplasia of the connective tissue elements, vascular sclerosis and degenerative atrophy of the muscle bundle.

With the progress of the lesion, the entire spindle is so altered in form and consistency that it is scarcely recognizable. The capsule is easily torn by the most delicate manipulation during the teasing dissection; as the vicinity of the spindle is approached, the intramuscular nerve trunk loses its staining property and appears to terminate abruptly in a shapeless mass of loose connective tissue having little or no resemblance to the fusiform neuromuscular bundle. At first, I often discarded this kind of tissue as unpromising material in which to make further search for the spindle; continued practice in teasing fresh muscle and the difficulty with which the spindle was discovered in some of my earlier specimens led me to suspect that these nests of fibrous tissue, in which the nerve ended blindly, might not be mere accumulations of ordinary endomysial connective tissue.

Microscopic examination of all material of this character disclosed, in many instances, unmistakable evidence of a spindle capsule; a compound spindle discovered in one of these fibrous masses is shown in the photomicrograph, Figure 18. The capsules of the two specimens are united in the equatorial region, and each preserves an arrangement suggestive of its original fusiform outline. Within the capsular remnants are many scattered fragments of degenerated intrafusal muscle, but there is not the slightest evidence of nerve fibers. Since spindles of this character were not discovered until late in the course of my study, the preserved Sihler material from six of my earlier cases was reexamined for additional evidence of this type of lesion and, by this means, ten spindles in this stage of the pathologic process were added to the collection.

That these accumulations of fibrous tissue are actually the remains of a disintegrated spindle is evident in the illustration of a second specimen of this character obtained from the same patient. The drawing, Figure 19, is partly diagrammatic; the capsular fibers have been omitted so that the contents of the spindle may be more easily recognized. Occasional fragments of intrafusal muscle are intermingled with an accumulation of nuclear material which is disposed in rows or chains within the muscle substance or as aggregations of nuclei that entirely replace the muscular tissue and resemble, in the lower magnification, abscess or tumor formations. It is apparent, however, in the enlarged segment of the spindle, represented in the lower drawing, that the cellular elements are derived from replacement hyperplasia of the sarcolemma nuclei. There is no evidence of an acute inflammation; the nerve tissue is totally destroyed, and, although the muscle bundle is completely disintegrated, the striations are still apparent in many of the fragments.

Finally, the entire contents of the spindle undergo fatty degeneration, and this stage of the lesion is represented in Figure 20. The specimen was first stained by the Sihler method, but this revealed no evidence of nerve or muscle;

the teased spindle was then stained with Scharlach R when the character of the lesion was disclosed. The capsular tissue and the general configuration of the spindle are still recognizable; the equatorial region is distended and filled with a mass of fat corpuscles; globules of fat are scattered throughout the specimen. These products of dissolution are later absorbed and replaced by a capsular scar enclosing a clear elongated or circular space which is distinguishable only in a series of transverse or longitudinal sections of the muscular tissue.

THE MUSCLE SPINDLE IN THE POSTENCEPHALITIC PARKINSONIAN SYNDROME

Because of the assertion that the changes described in the striated musculature in the parkinsonian disease are secondary to the tremor and rigidity, the lesions in the spindle might also be regarded as secondary to these continued variations in muscle tone. It is important, therefore, to know something about the condition of the neuromuscular bundle in other disorders associated with tremor and rigidity. I have accordingly examined a number of spindles from a case of the postencephalitic parkinsonian syndrome in which the muscle was obtained under conditions similar to those in the cases of the true parkinsonian affection.

W. W., a man, aged 57, was admitted to the Johns Hopkins Hospital Dispensary, Nov. 26, 1923, complaining of "paralysis and difficulty in making movements." He had had the usual diseases of early childhood and malaria at the age of 15, but enjoyed "excellent" health thereafter until 1918, when he suffered from "sleeping sickness." At that time he saw double and was alternately delirious and somnolent for a period of about four weeks. Within the second month of the illness, although still confined to bed, his general condition improved and the diplopia disappeared. Later, when he attempted to walk he could not "use" himself because all movements were stiff and clumsy. This stiffness was particularly noticeable in the arms. After a further rest of two or three months he made an effort to resume his occupation and continued to do "odd jobs" until July, 1922, when he was forced to discontinue all occupation because of great stiffness in all extremities. Tremor then appeared in both upper extremities and still persisted at the time of admission; it was of greater intensity on the right side and was always intensified in the performance of voluntary movements. The legs had not been tremulous, but the stiffness in them had so increased that he had great difficulty in arising from a chair without assistance.

The patient was admitted to the Church Home and Infirmary Nov. 27, 1923, where further history gave no evidence of syphilitic infection. Wassermann tests of the blood and spinal fluid were negative. On examination, all movements were slow and deliberate; the facial expression was masklike; speech was hesitating; associated movements of the extremities were abolished, and rigidity of the entire body was intense. Both during rest and on voluntary movement, a fine tremor was occasionally observed in the right upper extremity.

On the day of admission, Dr. Panettiere removed from the right abductor pollicis brevis a specimen of muscle, which was immediately placed in the usual fixing and staining solutions.

Four spindles were obtained from the teased material treated by the Sihler and the two gold chloride methods. All of the specimens are well stained, and none of them shows any evidence of swelling or changes in the intrafusal nerve or muscle. In the embedded material, three spindles are found in the cross sections and one is apparent in the longitudinal sections; but these sections reveal no abnormality in the spindle or in the extrafusal musculature. The

intramuscular blood vessels are moderately thickened. A cross section containing a double spindle, stained with hemalum and van Gieson stain, is represented in the photomicrograph, Figure 21. The greatest diameter of the two spindles is not more than 0.140 mm.; the intrafusal muscle is unaltered, and the capsule, although apparently thickened and exhibiting slight separation of the lamellae, has a width of only 0.014 mm. The intrafusal nerve fibers stain well with the van Gieson, the Weigert and the Bielschowsky methods. Although this study of the muscular tissue in a single case of the encephalitic affection does not warrant the conclusion that a peripheral lesion similar to that in paralysis agitans might not occur in the former disease, it is evident that in this instance tremor and rigidity of long duration were not productive of a lesion in the spindle, and that these symptoms should not, therefore, be designated as the cause of the changes occurring in the spindle in geuniue paralysis agitans.

SUMMARY AND COMMENT

Examination of the muscular tissue obtained by biopsy from fourteen cases of paralysis agitans and from one case of the postencephalitic parkinsonian syndrome reveals, in paralysis agitans, a lesion of the neuromuscular bundle which has not been previously described in this or in any other disease. The extrafusal musculature is unaltered, as a rule, and the intramuscular nerve trunks, except in the immediate vicinity of the spindle capsule, are of healthy appearance. With the exception of one instance in which the tissue was removed from the musculature of the forearm, the specimen was secured in each case from the abductor pollicis brevis and studied both in teased and in sectioned material. A total of ninety-six spindles was observed from the entire material; seventy-seven were found in the tissue from the cases of paralysis agitans; eight were derived from the muscle of the postencephalitic parkinsonian patient, and eleven were discovered in a specimen of healthy muscle.

The lesion in the muscle spindle is progressive, and is characterized by edema of the periaxial and capsular spaces, degeneration of the intrafusal nerve fibers, vascular sclerosis, hyperplasia of the connective tissue elements and disintegration of the muscle bundle. The entire contents of the spindle later undergo fatty changes, and are finally replaced by a connective tissue scar. In no instance is there evidence of an acute inflammatory reaction. The intrafusal muscle appears to be especially resistant to damage, since its normal structure is often preserved when other elements of the spindle are well advanced in the pathologic process. Although no special study was made of the pacinian bodies or the neurotendinous end-organs, these structures were found in several of the specimens of muscle, and they, as well as the motor end-plates, seemed to be unaltered. The peripheral lesion is, accordingly, confined to the neuromuscular bundle and appears to be essentially a disease of the nervous system affecting a specific system of proprioceptive fibers.

Reference has been made elsewhere to certain peculiarities in the character and distribution of the lesion. It does not necessarily involve all of the spindles. It is not advanced ordinarily to the same degree in every affected spindle, and the progress of the lesion appears to be correlated with the predominance of tremor or rigidity in the clinical syndrome. There is, however, no convincing anatomic evidence that the lesion in the spindle is the product of these clinical manifestations. Were tremor the cause of the changes in the spindle, the progress of the lesion should be proportionate to the intensity and duration of the symptom, but this hypothesis finds no confirmation in the pathologic material. Although fewer diseased spindles are found in the early course of the tremulous form of paralysis agitans than in the later stages of the affection, the spindle lesion is predominantly that of fibrosis, and this character is maintained so long as tremor is the outstanding symptom. It is also evident that in the rigid form of the affection, the number of diseased spindles likewise varies according to the duration of the illness, but the prevailing lesion in the spindle is invariably that of degeneration. It is not surprising, therefore, that in the classical parkinsonian disease in which both tremor and rigidity are prominent symptoms, many excellent specimens of the spindle are found in both stages of the pathologic process.

Since tremor rarely occurs without a certain amount of hypertonia, it has been asserted that rigidity and not tremor is the essential feature of paralysis agitans; and rigidity might, therefore, be regarded as the cause of the lesion in the muscle spindle. Nevertheless, the examination of eight spindles from a case of postencephalitic affection, in which stiffness was the dominant symptom for a number of years, failed to reveal any change in the neuromuscular bundle. Furthermore, it is difficult to understand why a structure so resistant as the spindle should exhibit this particular vulnerability to the mechanical effects of tremor or hypertonia while the surrounding musculature escapes uninjured, although it suffers in a variety of conditions in which the spindle preserves its integrity. Is not the spindle subjected to the repeated mechanical stimulation of muscular contraction throughout the active life of every artisan, and would not the continued action of the hypertonic or tremulous muscle tend to compress rather than distend the neuromuscular bundle?

If, then, these mechanical agencies are not responsible for the changes in the spindle, to what may they be attributed? The character of the lesion resembles that occurring in the reaction of tissue to some form of injury; in this instance, however, it is of chronic progressive nature, without evidence of an acute inflammatory process, and suggestive of the changes resulting from irritation by a long-continued, slowly acting agent. Chemical and bacterial toxins, as well as mechanical irritation,

are recognized factors in the production of an edematous or chronic inflammatory reaction, and the character of the spindle lesion suggests that it might depend on the selective action of a toxic substance on the nerve terminals in the neuromuscular bundle. Although there is no indication of the nature of the toxin or that it is specific in its action, this hypothesis is not out of harmony with that in a number of peripheral and central nervous affections. It is not unlikely that a variety of toxins might, under suitable conditions, exhibit a particular affinity for the nerve fibers of the muscle spindle; it is suggested that valuable information as to the nature of the irritant substance might be derived from a chemical study of the muscular tissue and the endocrine bodies. It is noteworthy that the parkinsonian symptoms occasionally appear during or shortly after an acute infectious illness, and significance seems to attach to the changes in the intrafusal muscle in phosphorus poisoning and in exophthalmic goiter.

MUSCLE TONE AND THE MUSCLE SPINDLES

If the lesion in the neuromuscular bundle is not secondary to the tremor or the rigidity but is an essential part of the pathology of paralysis agitans, it is imperative that its significance in the clinical syndrome be determined; this involves necessarily a consideration of some of the features of muscle tone and the function of the muscle spindle.

Fundamentally, the production, recognition and maintenance of normal muscle tone depend on unimpaired axonal function in the anterior and posterior spinal roots, but it has been asserted that muscle tone is a "proprioceptive reflex whose source is in the tonic muscle itself." This is no more than a statement that muscle tone is a tone reflex originating in a tonic muscle. If tone be a proprioceptive reflex originating in the muscle, by what means is the afferent component of this reflex conveyed to the central nervous system? Since the motor end-plate and the sympathetic nerve-endings are the only nerve terminals in the muscle tissue proper, it is obvious that the autonomic nervous system alone must subserve this purpose if the afferent impulse originates in the muscle cell. If, however, the tone mechanism is provided with a specific afferent axon, is this function to be assigned to the fibers terminating in the pacinian body, in the neurotendinous end-organ or in the muscle spindle? It is more than likely that each of these specialized organs, as well as the stimuli from a variety of afferent axons, has its effect on muscle tone. An intricate central tone mechanism, with cortical inhibition and basilar integration, protects the motor neuron from conflicting reflex stimulation. It is to this central nuclear region that the attention of neurologists has been directed; and, although the thalamic connections of these nuclei bring many of their neurons into direct relation with a variety of afferent

impulses, the studies of this region have been largely concerned with the efferent motor system of fibers.

The thalamus, however, is the central nucleus for the reception of many afferent impressions; it possesses a cortical system of fibers which conducts impulses in both directions; an interchange of axons is also established with the corpus striatum and the subthalamic nuclei; but these nuclear structures have no direct cortical communication and are largely under the control of their thalamic connections. What, then, is the inhibitory mechanism provided for the afferent impulses terminating in the thalamus? Is it entirely cortical, or might not there be, in the afferent system itself, a proprioceptive inhibitory mechanism whose function it is to reduce to a common level the various sensory impulses which, through their thalamic terminations, might reflexly alter the tonic state of the muscle? Since there is much in the study of my material to suggest that the muscle spindle plays a very definite part in the mechanism of muscle tone, I am of the opinion that it is the proprioceptive organ through which this afferent inhibition might be exerted; but the neuromuscular bundle is said to be an organ of muscle sense, and yet this sense is not disturbed in paralysis agitans although the spindle is obviously diseased.

Although nothing is known of the central course or termination of the axons from the neuromuscular bundle, it is not unlikely that they have a disposition analogous to those of the afferent system in general. Presumably, many of them terminate, through neurons of the second order, in the thalamic nuclei where, it is assumed, the impulse from the neuromuscular bundle inhibits or modifies the various afferent stimuli concerned in the reflex mechanism of muscle tone. If this hypothesis is correct, it is apparent that a disturbance in muscle tone might result from a lesion of either the peripheral or the central ending of this inhibitory system, and that in the central lesion the symptomatology is likely to be more inclusive, more widely distributed at the outset and more variable in type than when the lesion makes its appearance at the periphery. This difference in the situation of the lesion is offered as a possible explanation of the dissimilarity of clinical manifestations in genuine paralysis agitans and the postencephalitic parkinsonian syndrome. Nevertheless, it is conceivable that the occasional combination of the central and the peripheral lesion might be responsible for the extraordinary clinical resemblance sometimes exhibited by the two affections.

It is furthermore possible, on this hypothesis, to find within the diseased spindle an explanation of the two types of motor disturbance characteristic of the parkinsonian affection. Rigidity and tremor are said to be only different manifestations of a fundamental disorder of muscle tone and, although rigidity may not be "tremor run together," it

is apparent that alternate rhythmic increment and decrement in the tension of a muscle are essential physical changes in the development of tremor. Reciprocal stimulation and inhibition are therefore necessary to the production of these changes in the physical state of the muscle, and either the withdrawal of inhibition or the intensification of the motor stimulus might result in the appearance of hypertonic rigidity. It is accordingly difficult to conceive of the neuromuscular bundle as the source of a proprioceptive impulse which serves to activate the motor component of the tone reflex, or else destruction of the spindle could take no part in the production of tremor or rigidity; but as an inhibitory organ its significance in the pathology of the disease is more readily understood.

Thus, in the early fibrosed stage of the lesion the inhibitory influence of the spindle is only partially affected and, as a consequence, afferent stimulation of the tonectic centers is augmented and irregular. A tremor of repose, subjugated by the inhibition of volitional movement, and a disturbance of automatic acts are the result. With complete and widespread destruction of the spindle and progressive extinction of peripheral inhibition, an afferent reflex rigidity of the extrapyramidal type makes its appearance.

This hypothetic function of the spindle and the interpretation of the morbid process find an apparent contradiction in the fact that disease of the neuromuscular bundle has been observed also after complete lesions of the peripheral nerves, in the myopathies, and to a questionable degree in tabes dorsalis; but tremor—unless the incoordination of tabes is a form of tremor—and hypertonia do not appear in the symptomatology of these disorders. It is obvious, however, that in these diseases the lesion is not confined to the muscle spindle. All impulses are abolished in a complete nerve lesion; in the myopathies no lesion of the spindle is observed unless the surrounding musculature is so altered that the development of a tremulous or hypertonic response is not possible; and in tabes, even though the spindle nerves might be affected, the lesion is not confined to these axons, nor would disease of the spindle be apparent unless the syphilitic process had invaded the spinal ganglion or the distal portion of the posterior root.

The recent studies of Walshe,[30] in which he attempts to "de-afferent" the parkinsonian muscle by the deep injection of a 1 per cent. solution of procaine into the motor point of the biceps brachii, appear also to discredit my hypothesis concerning the nature and function of the muscle spindle. Following the injection of small quantities of the solution, he observed no appreciable reduction of the tremor or rigidity but, if larger

30. Walshe, F. M. R.: Observations on the Nature of the Muscular Rigidity of Paralysis Agitans, and on Its Relationship to Tremor, Brain **47**:159 (May) 1924.

quantities were used, the rigidity was entirely abolished while the tremor retained its original intensity and was, under certain circumstances, apparently increased. With the injection of still larger quantities of the solution, all reflex and voluntary movement of the muscle was destroyed, and Walshe admits that this extensive paralysis might have been due to the action of the drug on the motor nerve terminals or on the muscle tissue itself.

If, however, the action of the solution in smaller quantities is purely selective and confined to the afferent nerve terminals, what assurance is there that all of these nerve endings in the biceps muscle are equally affected; or that the proprioceptive terminals in the coracobrachialis and the brachialis anticus, innervated by the same nerve as the biceps, are also affected; or that the exteroceptive impulses from the overlying cutaneous area cease to function, and that these axons do not, therefore, convey afferent reflex stimuli to the lower motor neurons or to the tone centers of the injected muscle? Apparently it is not possible, by means of a local injection, to "de-afferent", completely the spinal center of a muscle or muscle group; and, unless the motor center of the injected muscle is protected from the collateral afferent impulses conveyed through the common nerve trunk, it is unlikely that both tremor and rigidity will be entirely abolished. In paralysis agitans, posterior rhizotomy, unless widely practiced, is probably no more effective, and Walshe refers to the inconclusive results obtained by Leriche in the performance of this operation.

It seems, therefore, that in the experiments conducted by Walshe, there has been secured an appreciable reduction in the number of afferent impulses originating in the injected muscle and, with its afferent tone mechanism thus reduced to a lower level, the hypertonic rigidity is necessarily diminished; but the muscle is not completely atonic or else voluntary movement must also be abolished. The lower motor neuron of the de-afferented muscle is unaffected and is still subjected to the disorganized peripheral and central reflex irritation consequent on disease of the spindle in the uninjected musculature innervated from the same spinal segment; although this collateral stimulation might not be adequate for the production of rigidity, it is offered as a possible explanation for the preservation of tremor in the desensitized muscle. It seems, therefore, that the conditions of the experiment do not warrant Walshe's conclusion that "tremor is unaffected by de-afferenting the tremulous muscles and cannot, therefore, be a proprioceptive reflex."

CONCLUSIONS

1. Evidence has accumulated within the past decade which indicates that the postencephalitic parkinsonian syndrome and genuine paralysis agitans are often accompanied by a lesion in one or more of the basal

ganglionic structures; nevertheless, it has been quite impossible to determine the exact location of the lesion in either instance, and the pathologic and clinical identity of the two diseases is not yet universally recognized. There is much in the clinical course of paralysis agitans to suggest that in some instances, at least, it may be dependent on a morbid process situated in the peripheral mechanism concerned in the production and maintenance of muscle tone, and unsuccessful attempts have been made to locate the lesion in the muscle tissue itself. My examination of the muscular tissue obtained by biopsy from fourteen cases of genuine paralysis agitans gives no evidence that the disease is of myopathic origin.

2. There is, however, in the neuromuscular bundle a constant lesion characterized by edema, early degeneration of the intrafusal nerve and final disintegration of the entire spindle. The intramuscular nerve trunks are normal, and the surrounding musculature is, as a rule, unaltered. The lesion in the neuromuscular bundle is, therefore, essentially neuropathic and involves a specific system of proprioceptive nerve terminals.

3. That the lesion in the spindle is primary, and not secondary to the tremor or the rigidity, is indicated by the failure to discover any abnormality in the eight spindles obtained from a case of the postencephalitic parkinsonian syndrome in which these symptoms had persisted throughout a period of five years.

4. Although studied in a variety of diseases, no lesion similar to that observed in paralysis agitans has been discovered in the neuromuscular bundle. Because the spindle has not been examined in all cases under the same technical conditions, I cannot, however, make the unqualified assertion that the lesion is pathognomonic of paralysis agitans. Nevertheless, had a spindle lesion similar to that exhibited in my microscopic sections existed in any other disease, it could not well have escaped detection by the simpler technical methods there employed.

5. There is reason to believe that the lesion is a toxic parenchymatous degeneration of the intrafusal nerve terminals. The cause of the lesion is not known, but it is conceivable that one or more toxic substances of metabolic or glandular origin might exhibit a selective affinity for the neuromuscular bundle, and the early edematous change in the spindle suggests an irritant chemical action or osmotic process.

6. It is generally taught that the muscle spindle is an organ of muscle sense, but apparently disease of the neuromuscular bundle causes no appreciable abnormality in the perception of this sense. It is therefore reasonable to conclude that the spindle does not subserve this function. There is, however, much evidence that it plays an essential part in the

Fig. 1.—Normal spindle from T. L.; teased specimen; Sihler stain; camera lucida drawing; × 32. The intrafusal muscle and the capsule are apparent, and an equatorial nerve trunk, a polar nerve trunk to the left and the blood vessels are shown entering the capsule. The large nerve, between the equatorial and the polar nerves, contributes no fibers to the spindle.

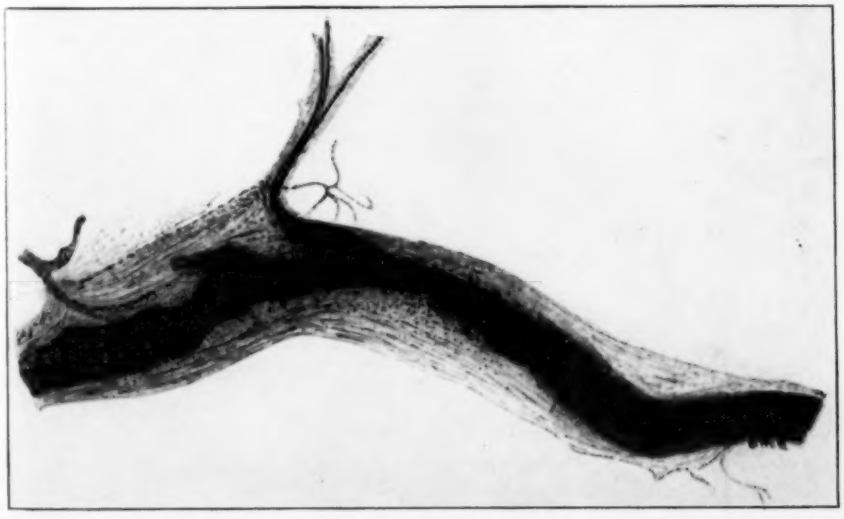


Fig. 2.—Higher magnification of the central portion of the spindle shown in Figure 1. Camera lucida drawing; × 70. The capsule, the equatorial nerve trunk and the intrafusal nerve fibers are distinctly visible, but the individual fibers of the muscle bundle are not apparent.

Fig. 3.—The left polar extremity of the spindle shown in Figure 1, × 700. Three intrafusal muscle fibers, their striation, nuclei and nerve terminals are visible. The large deeply staining nerve fiber above, derived from the intrafusal nerve plexus to the right, extends to the extremity of the spindle. Three smaller nerve fibers are apparent between the muscle fibers. The middle and the lower muscle fibers are partly covered by numerous nerve-endings.

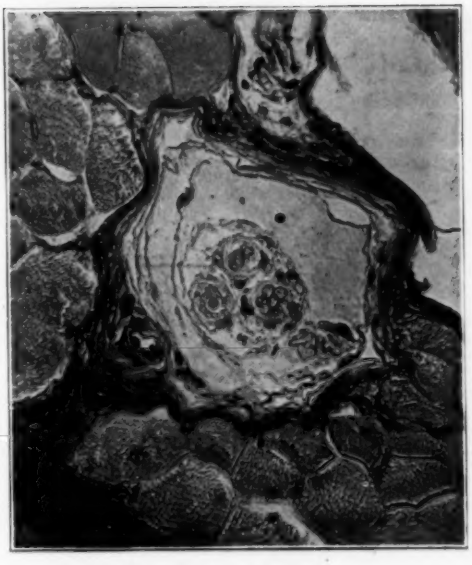


Fig. 4.—Photomicrograph, normal spindle from T. L. Zenker fixation; cross section, equatorial region; hematoxylin and van Gieson stain; × 280. The capsule, the periaxial space, the axial sheath, the muscle bundle and the extrafusal musculature may be noted. Actual dimensions of the spindle: diameter, 0.171 mm.; capsular thickness, 0.026 mm.; width of periaxial space, 0.033 mm.; average diameter of intrafusal muscle, 0.019 mm.

Fig. 5.—Higher magnification of the section shown in Figure 4. Photomicrograph, slightly retouched; × 630. The structure of the intrafusal muscle, the axial sheath and the absence of tissue in the periaxial space may be observed.

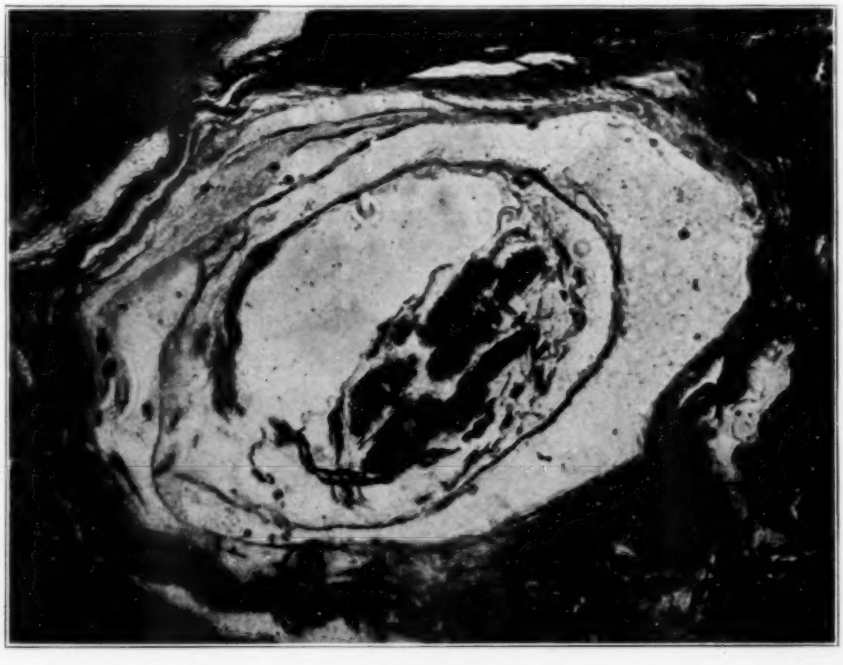


Fig. 6.—Photomicrograph, Bielschowsky preparation; normal spindle from T. L., longitudinal section; × 280. The specimen contains several healthy axis cylinders within the axial sheath and on the intrafusal muscle, in which the striations and the central nuclei are also apparent. The emptiness of the periaxial space may also be observed.

Fig. 7.—Longitudinal section, equatorial region, normal spindle, intercostal muscle of child; hematoxylin and eosin stain; camera lucida drawing; × 852. The delicate structure of the capsule and the axial sheath, the fine fibrillar strands extending across the periaxial space and the axons of the intrafusal nerve in the axial sheath just above the right extremity of the muscle bundle may be noted. Actual dimensions of the spindle: diameter, 0.108 mm.; length, 0.264 mm.; width of the periaxial space, 0.033 mm.; diameter of the axis cylinders, 0.0014 mm.

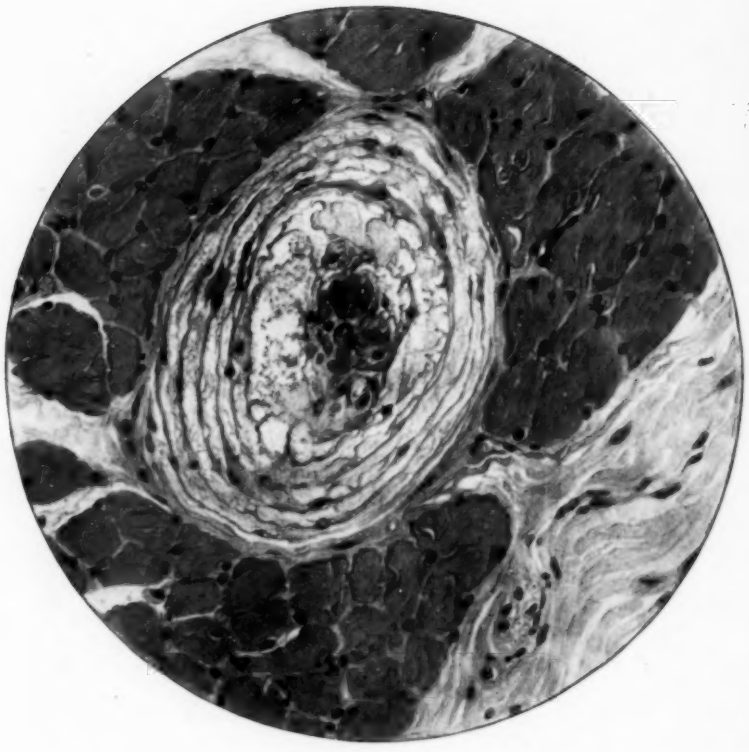


Fig. 8.—Cross section, equatorial region of a spindle from Case 1, showing the early lesion in paralysis agitans; hematoxylin and eosin stain; × 220. Comparison should be made with Figures 4, 5 and 7. The enlargement of the spindle, the distention of the capsular and periaxial spaces and the increase of connective tissue and nuclei in both these regions may be noted. The intrafusal muscle shows slight lacunar degeneration in two of the fibers. The extrafusal musculature is normal. Cross sections of healthy axons are visible in the endomysium below and to the right of the spindle. Dimensions: diameter, 0.283 mm.; capsular thickness, 0.068 mm.; width of periaxial space, 0.045 mm.; diameter of largest intrafusal muscle fibers, 0.018 mm.

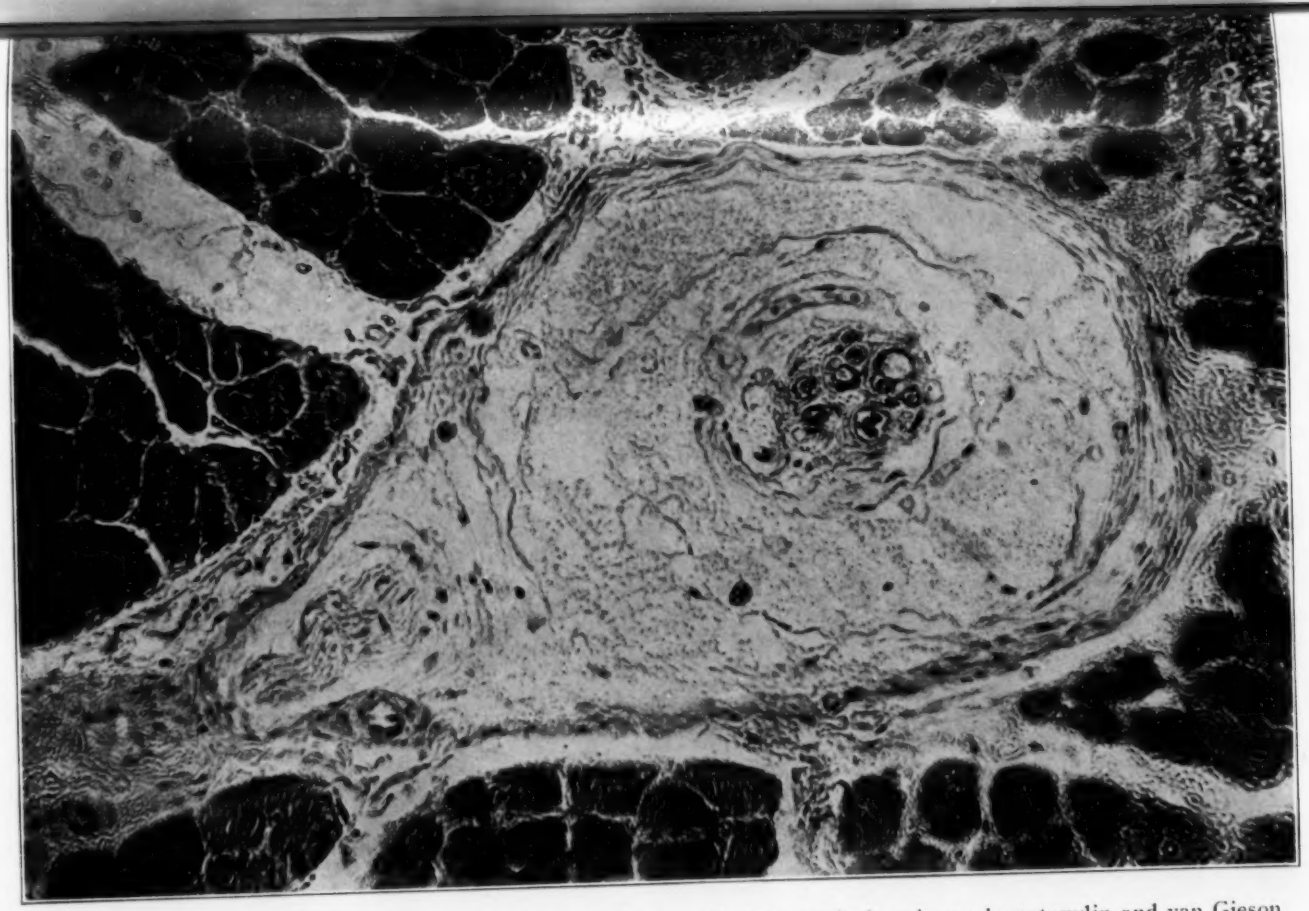


Fig. 9.—Cross section; equatorial region of a spindle from Case 2, paralysis agitans; hematoxylin and van Gieson stain; photomicrograph; × 280. The spindle is greatly swollen. Comparison should be made with the normal specimen, Figure 4, photographed at the same magnification. Partly degenerated nerve within the capsule at the extreme left, disintegration of muscle bundle, sclerosis of axial tissue and blood vessels and content of periaxial space will be noted. Dimensions: diameter, 0.488 mm.; capsular width, 0.035 mm.; width of the periaxial space, 0.107 mm.; maximum diameter of intrafusal muscle fiber, 0.017 mm.

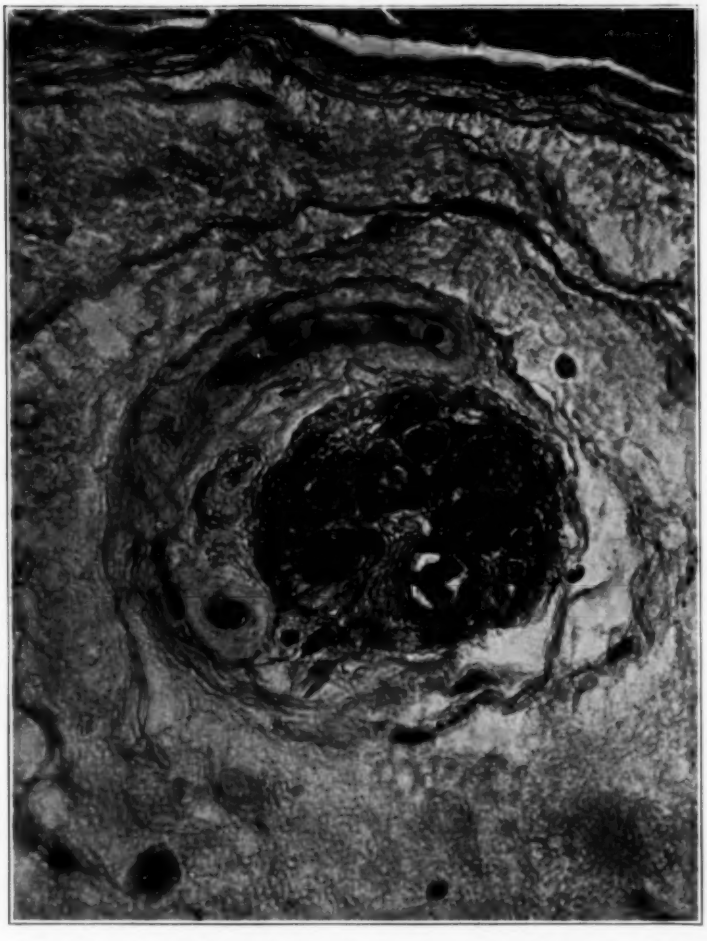


Fig. 10.—Central portion of the spindle shown in Figure 9. Photomicrograph; × 630. The disintegration of the intrafusal muscle, thickening of the axial sheath and blood vessels and sclerosis of tissue in the periaxial space may be observed. Comparison should be made with the normal spindle, Figure 5, photographed at the same magnification.

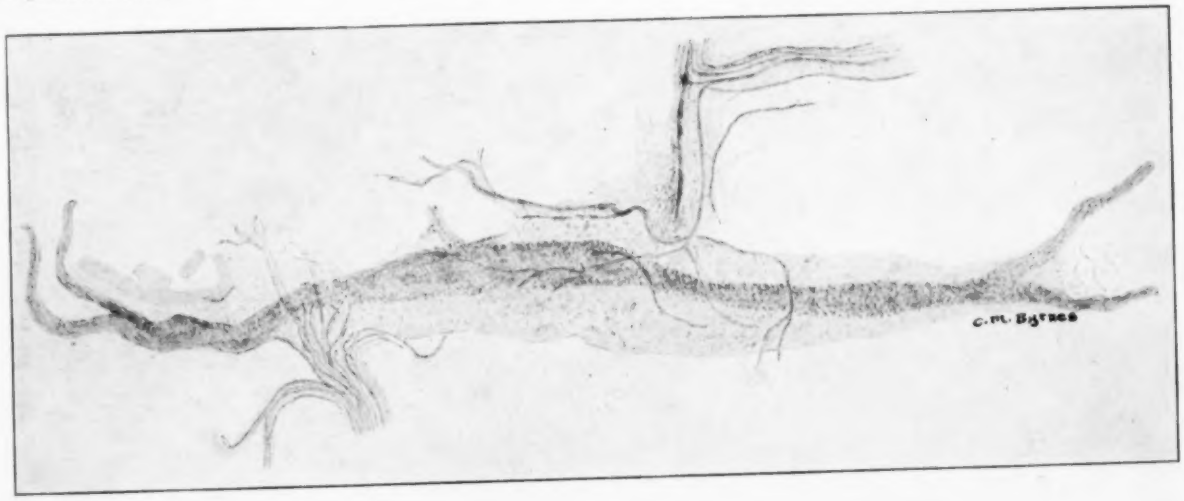
Fig. 11.—Photomicrograph of a teased spindle, Case 3, paralysis agitans; × 33; Ranvier gold chloride method. The capsule, the equatorial nerve trunk and vessels and the intrafusal muscle and nerve are evident. The distention of the capsule is the only apparent abnormality. Dimensions: length, 5 mm.; equatorial width, 0.714 mm.

Fig. 12.—Double spindle from Case 3, paralysis agitans; teased specimen; Ranvier gold chloride method; × 25. The nerves to both spindles are well stained, but in the upper specimen, the intrafusal fibers are not stained in the polar region. The capsule is distended at the equator. The lower spindle is normal. Dimensions: upper spindle: length, 6.1 mm.; equatorial diameter, 0.633 mm.; lower spindle: length, 5.3 mm.; equatorial diameter, 0.2 mm.

Fig. 13.—Portion of a spindle from Case 4, paralysis agitans; teased specimen; Ranvier gold method; × 47. The entire absence of nerve fibers within the spindle may be noted. The staining of the equatorial nerve trunk ceases just beyond the capsular margin.

Fig. 14.—Teased spindle from Case 4; paralysis agitans with great rigidity; × 32. The specimen was first treated by the Ranvier gold method, then counterstained by the Sihler method. The drawing is a composite picture of the doubly stained spindle. Description in text. Swelling at equator, nerve fragments in the capsule and deeply stained distal portion of the equatorial nerve trunk may be noted. Width at equator, 0.720 mm.

Fig. 15.—Teased spindle, Case 3; paralysis agitans; × 32; stained by Sihler method only. The entire spindle takes the stain faintly. Only one axon from the equatorial nerve trunk enters the spindle, the other ceases to stain at the capsular margin. The intrafusal nerve fibers stain faintly and only in the equatorial region. Comparison should be made with the normal spindle, Figure 2.



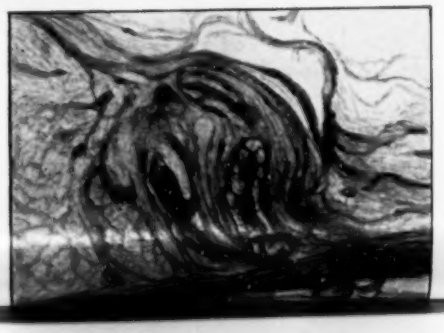


Fig. 16.—Longitudinal section, equatorial region of a spindle from Case 3, paralysis agitans. Tremor was the dominant symptom. Phosphotungstic acid-hematoxylin stain. (Description in text.) The size of the spindle, the intense fibrosis and the nerve and muscle degeneration should be noted. Drawing of spindle, × 173; insert, × 980. Dimensions: length, 1.155 mm.; diameter, 0.423 mm. The appearance of the axial sheath and the periaxial space should be compared with that of the normal spindle in Figure 7.

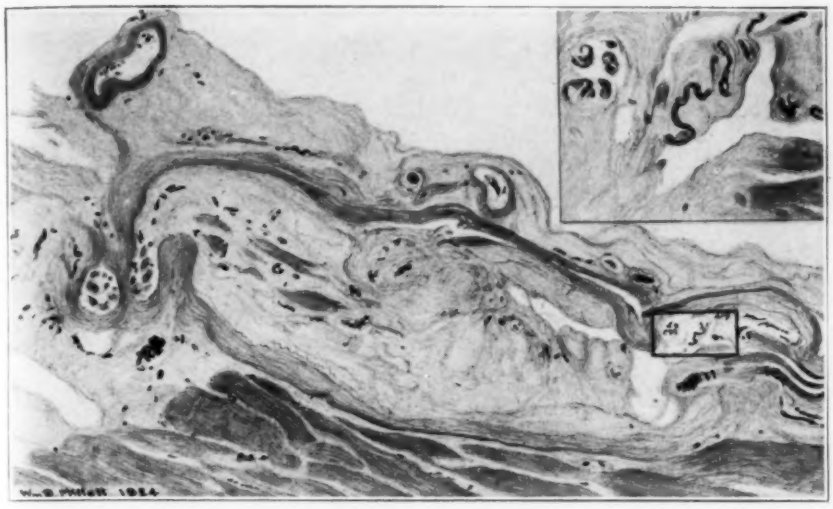


Fig. 17.—Longitudinal section of the same spindle as that shown in Figure 16. Weigert myelin stain. Almost total degeneration of intrafusal nerve. Disintegration of the myelin is apparent in the enlarged sketch of the rectangular area in the spindle. Extensive hyperplasia of the fibrous tissue. Many of the dark bodies in the periaxial tissue are sectioned blood vessels. Moderate vascular sclerosis.

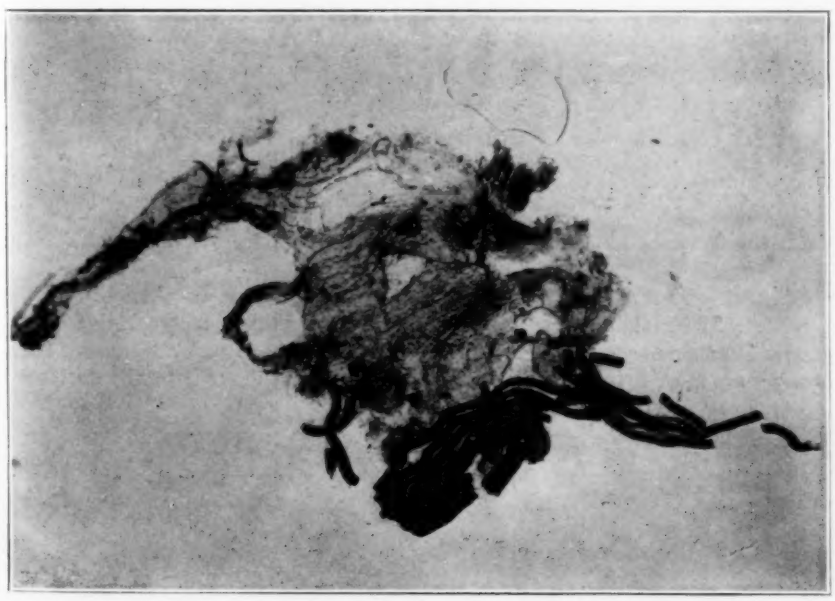


Fig. 18.—Photomicrograph, × 20, of a greatly disintegrated compound spindle from Case 4, paralysis agitans; rigidity was the dominant symptom. Teased specimen; Sihler stain. This magnification reveals only the disorganized capsular tissue.

Fig. 19.—Partly diagrammatic sketch of a spindle from Case 4. Teased specimen, Sihler stain. The capsular fibers are omitted. Degeneration of the muscle bundle and proliferation of sarcolemma nuclei are well advanced. These changes are more apparent in the lower drawing. The nerve is totally destroyed.

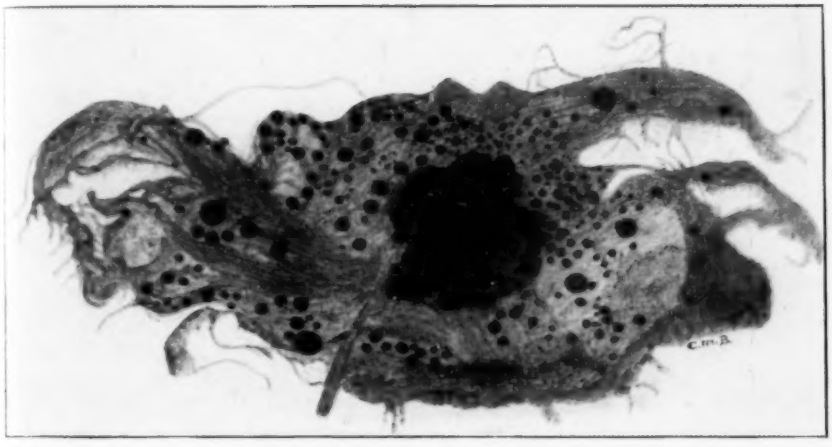


Fig. 20.—Muscle spindle, Case 3, paralysis agitans. Teased specimen from the Sihler material counterstained with Scharlach R, showing the final stage of the lesion in the spindle. The capsular remnant is filled with globules of fat and contains no evidence of nerve or muscle tissue.

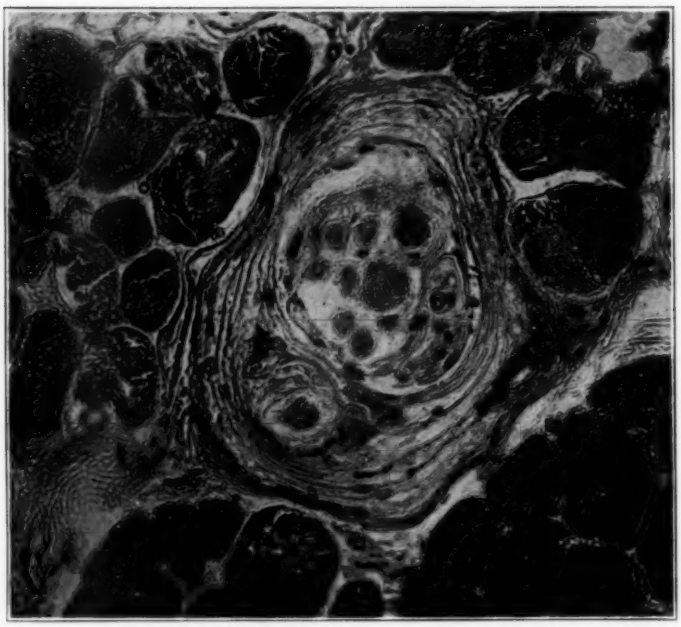


Fig. 21.—Cross section of a double spindle from postencephalitic parkinsonian syndrome. Photomicrograph, × 280. Hemalum and van Gieson stain. The intrafusal blood vessel is slightly thickened, but the spindles are otherwise normal. Dimensions: diameter of the larger spindle, 0.140 mm.; capsular thickness, 0.014 mm. Comparison should be made with Figures 4, 9 and 10.

mechanism of muscle tone, and it is my belief that the spindle is the afferent inhibitor of the striothalamic tonectic centers.

7. Hypertonia of the parkinsonian type might originate, therefore, from either central or peripheral disease of this system of fibers; and it is suggested that the central lesion finds its chief exponent in the post-encephalitic syndrome, while genuine paralysis agitans, in its typical form, is characteristic of the peripheral lesion. This assumption does not preclude, however, the possibility of a combination of the two lesions in certain types of Parkinson's disease in which the symptoms are widespread and fully developed in the early course of the disorder.

207 East Preston Street.

SENSORY DISTURBANCES IN TUMORS OF THE CERVICAL SPINAL CORD

ARRANGEMENT OF THE FIBERS IN THE SENSORY PATHWAYS *

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AND

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The symptoms and signs of disturbed motility and of altered sensibility, observed in many patients with compression of the spinal cord by a neoplasm, form a syndrome which is typical of pressure on the spinal cord. In not a few instances, however, the disturbance of motor power is much more advanced than that of sensation, and often the diminution of one or other type of sensibility is out of proportion to the changes in other modalities. The study of these clinical variations, which may be found in tumors at all levels but are often striking in compression of the cervical segments of the spinal cord, not only has yielded valuable information for the diagnosis and differential diagnosis of spinal disease, but also has advanced our knowledge of the course and the arrangement of the fibers in the sensory pathways.

In extramedullary tumors, it is often possible to obtain the history that the subjective sensory disturbances began in the most distal parts of the lower extremities and gradually advanced in a cephalad direction; and, on the other hand, in intramedullary growths, that diminished sensibility was first observed on the chest or abdomen and then spread in a caudad direction. The same order of progression may be found if the patient has been under observation for a long period during which repeated sensory examinations have been made. We have also observed it in persons in whom the sensory and motor disturbances had become aggravated after the withdrawal of fluid by lumbar puncture.

A difference in the power of resistance to pressure of the different fibers is not an adequate explanation for these progressive changes. It is much more probable that in the spinothalamic tracts there is a lamellar arrangement of fibers, according to limbs and the different parts of the limbs and trunk.

The arrangement in the lateral spinothalamic tracts is such that the fibers which carry sensation from the most caudal dermatomes lie in the external and posterior part of each pathway, while the fibers from

* Read before the American Neurological Association, Washington, D. C., May 5, 1925.

higher skin areas are successively placed nearer to the central gray matter of the cord, more and more internal and anterior to fibers from lower dermatomes (Fig. 4).

In the early stages of compression of the spinal cord by an extramedullary tumor which lies dorsally or dorsolaterally, only the most external and posterior parts of the lateral spinothalamic tracts may be affected by the pressure; and disturbances of sensibility may be found only in dermatomes well below the actual segmentary level of the lesion (Fig. 1). If all cutaneous sensibility has been disturbed below the

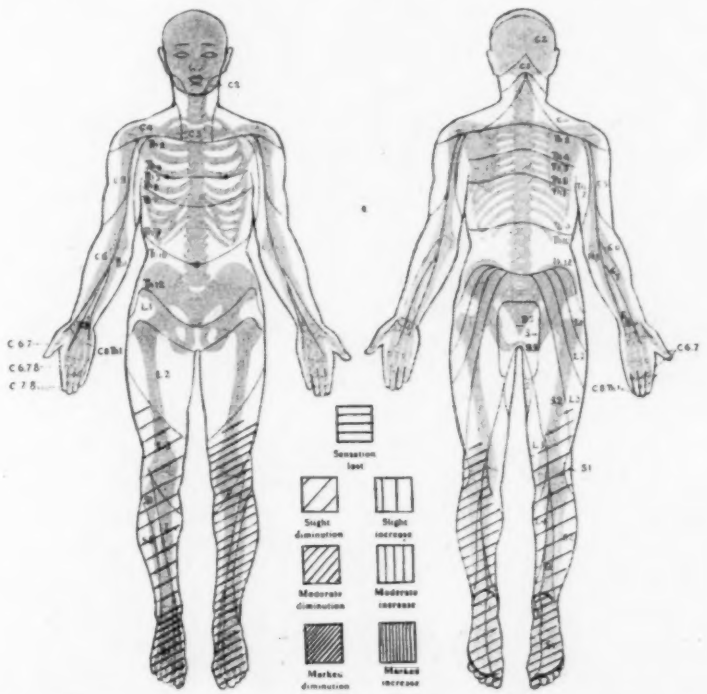

Fig. 1.—Cutaneous sensory disturbances in a case of extramedullary tumor at the twelfth thoracic segment, showing the absence of sensory disturbance above the third lumbar dermatome.

segment compressed by the new growth, the greatest diminution of sensation will often be found in the most caudal dermatomic areas (Fig. 2).

In the early stages of cord compression by an extramedullary tumor on the median ventral aspect of the cord, sensibility is often undisturbed in the most caudal dermatomes (Fig. 3); which means that, at first, the pressure of the expanding lesion was transmitted only to the most anterior and internal parts of the lateral spinothalamic tracts. On the

other hand, in pressure on the cord from behind by a tumor in or near the midline, such a relative sparing of fibers from the more caudal skin areas is never seen.

Head, Karplus, Babinski, Barré and Jarkowski, Thomas and Foerster have described cases of extramedullary cord compression in which the sacral dermatomes were not involved in the sensory disturbances. Collins and Marks mentioned that medullary sensory disturbances may appear in regions far removed from the site of a spinal cord tumor. Roux-Paviot published a case of extramedullary growth at the level of the

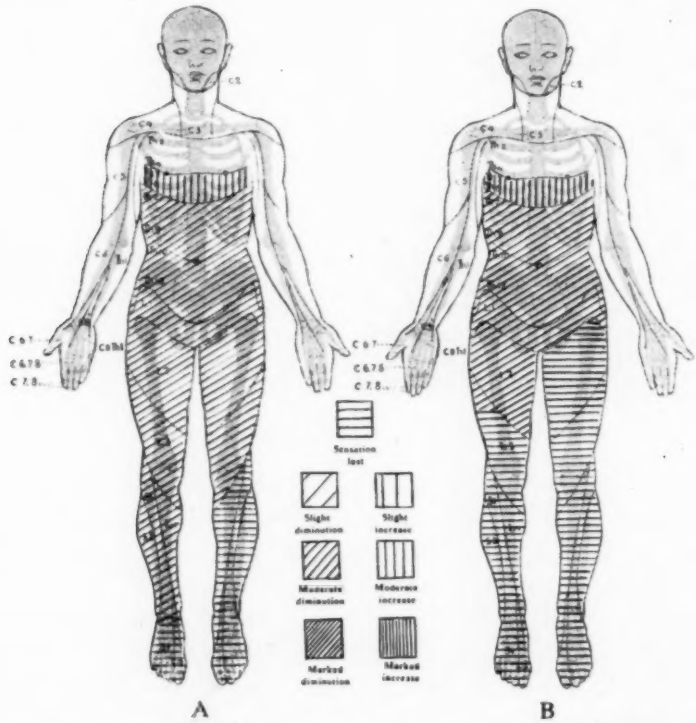

Fig. 2.—Cutaneous sensory disturbance in a patient with a tumor at the fifth and sixth thoracic segments, showing that the greatest diminution of sensibility is in the most caudal dermatomes; *A*, tactile; *B*, pain and temperature.

lower cervical and upper thoracic segments, in which sensation was lost only in both feet. Serko, in a paper on spinal cord tumors, states that in extramedullary compression sensation becomes diminished in an ascending direction, and returns, after the removal of the tumor, in a descending direction.

A number of cases have been reported in which, after the division of anterolateral tracts of the cord for pain, the upper level of loss of pain and temperature sensibility occurred in dermatomes far below the

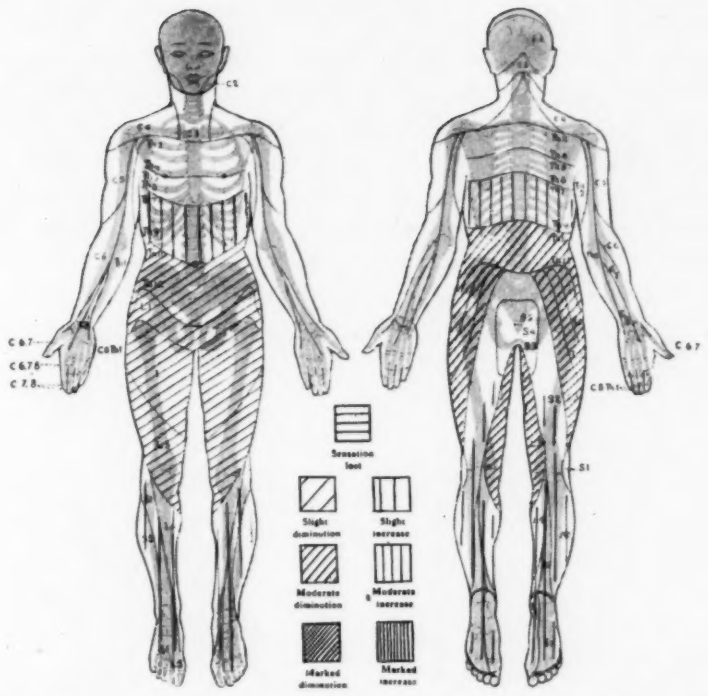

Fig. 3.—Cutaneous sensory disturbances in a patient with an extramedullary tumor on the anterior aspect of the cord. It may be noted that sensation is preserved in the caudal sacral dermatomes.

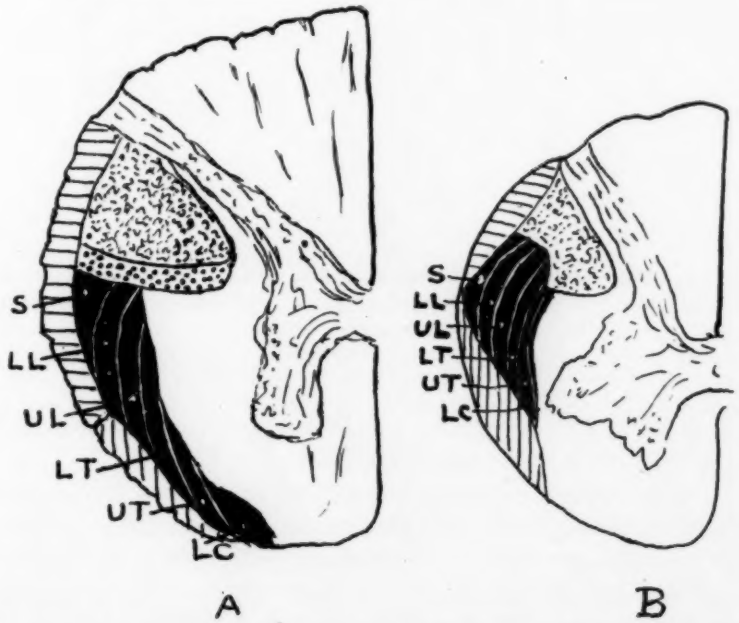

Fig. 4.—The lamellar arrangement of the fibers in the lateral spinothalamic tracts. *S*, sacral; *LL*, lower lumbar; *UL*, upper lumbar; *LT*, lower thoracic; *UT*, upper thoracic; *LC*, lower cervical. The location and size of the spinothalamic tract in *A* is from Tilney and Riley, and in *B* from Dejerine.

segment at which the cord had been incised. This might be explained in part by the obliquity of crossing of the fibers which carry these types of sensibility, but it is more probable that the incision in the cord was not deep enough to divide the innermost and most anterior parts of the spinothalamic tracts.

All of these published reports support our own views that in the spinothalamic pathways the fibers which convey sensation from the most caudal skin areas lie in the posterior and outer part of each tract,

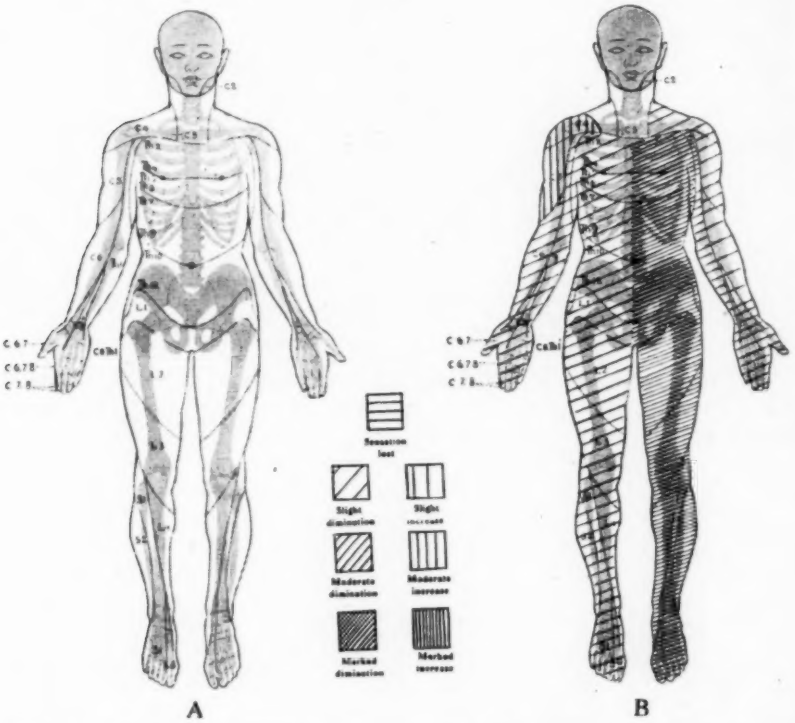

Fig. 5.—Cutaneous sensory disturbances in a patient (C. S.) with an extramedullary tumor on the right ventrolateral aspect of the fourth and fifth cervical segments. It may be noted that tactile sensibility is normal, while pain and temperature are markedly diminished; *A*, tactile; *B*, pain and temperature.

and those which join at higher levels take a position somewhat anterior and internal to the ones already in the tract (Fig. 4).

It is well known that, in the posterior white columns which transmit the so-called "deep" sensibilities upward on the same side of the cord, there is a lamellar arrangement of the fibers. The fibers which enter the cord at each higher level take a position external and somewhat posterior to those that have already entered the cord, so that those from the most caudal dermatomes finally lie nearest the posterior median fissure. As

the fibers that finally run in the spinothalamic tracts cross over to the opposite side of the cord before they enter the tracts, it is natural that the fibers from higher levels should lie mesial to those which have already entered the pathways in the lateral white columns of the cord.

In extramedullary compression of the spinal cord, and especially in cervical cord tumors, there is often a marked dissociation between the different modalities of cutaneous sensibility. Unless the patient comes

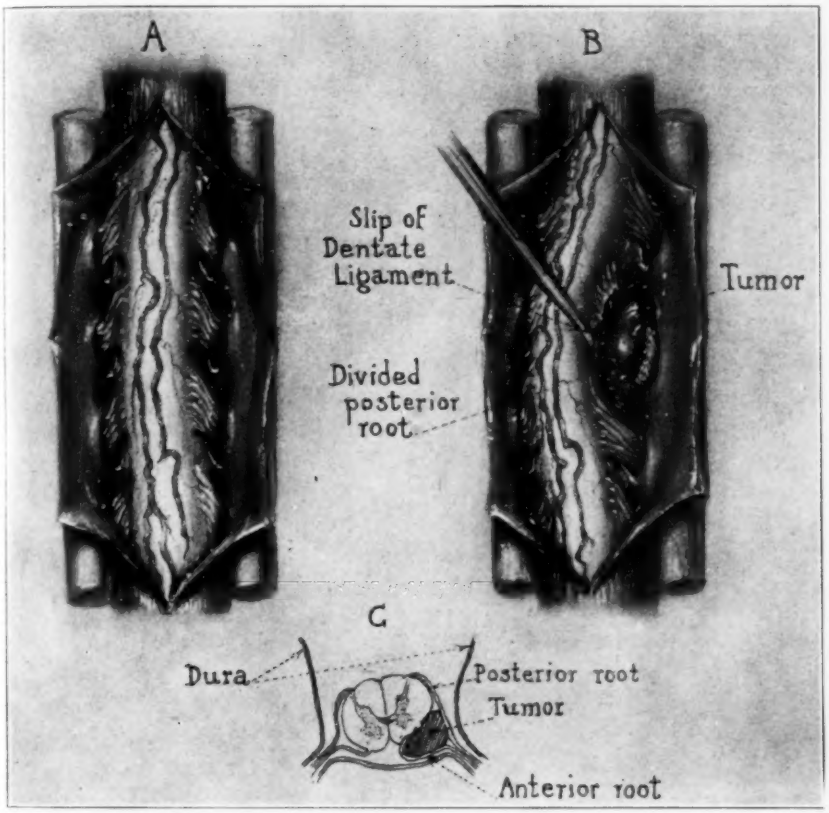

Fig. 6.—Condition found at operation in patient (C. S.) whose sensory disturbances are shown in Figure 5. *A*, appearance of the cord at operation; *B*, the tumor exposed; *C*, the location of the growth. (From Surgical Clinics of North America, 5, No. 1, 1925, W. B. Saunders Company, Philadelphia.)

under observation when all cutaneous sensibility has been lost, tactile sensibility is often very little or not all affected (Figs. 5 and 6).

This dissociation between disturbances of the ability to recognize the light touch of cotton on the one hand, and difference of temperature and the pain of a pin prick on the other, has always been considered to be characteristic of intramedullary disease of the spinal cord, but this

so-called syringomyelic dissociation is often observed in extramedullary cervical cord tumors.

If, as has been generally accepted, the fibers for tactile sense run in several pathways, the frequent dissociation between tactile and the other cutaneous sensibilities, can, perhaps, be partially explained on that basis.

The frequency of the relative sparing of the tactile compared with the other cutaneous sensibilities in ventrally placed growths has led us to suspect that the greater number of the fibers which convey the feeling of touch run in pathways in the posterior white columns and the most posterior parts of the lateral white columns of the cord.

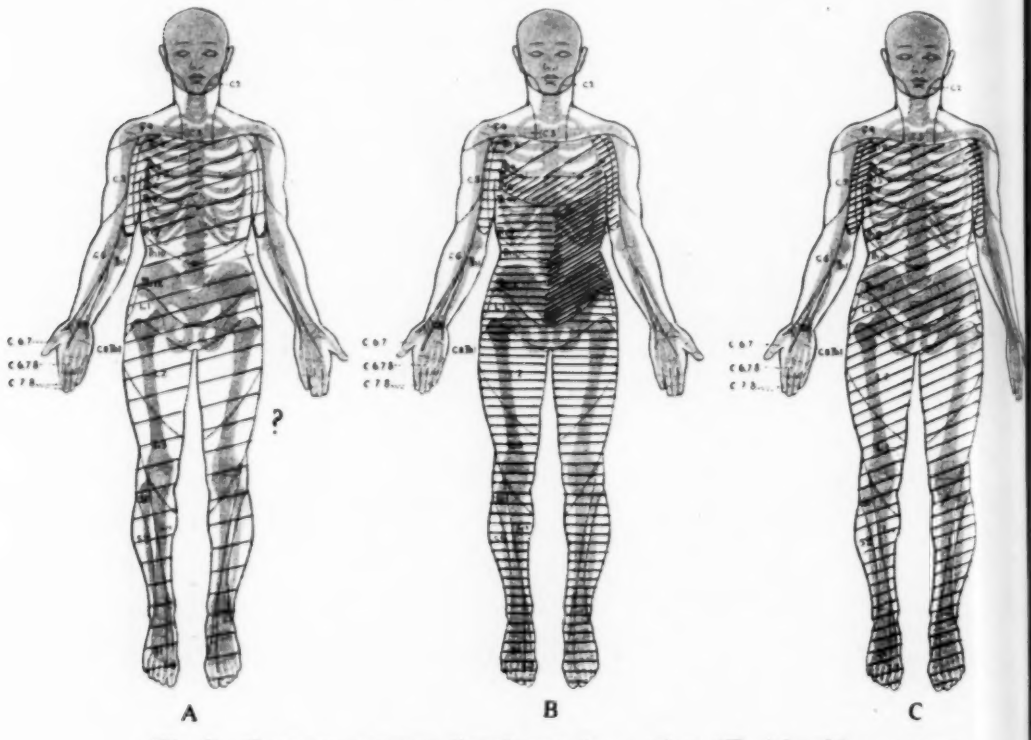

Fig. 7.—Cutaneous sensory disturbances in a patient (T. A.) with an extramedullary tumor on the left ventrolateral aspect of the eighth cervical and first thoracic segments. Tactile sensibility little, if at all, diminished; pain lost over a large part of the body over the area in which temperature was only moderately diminished; *A*, tactile; *B*, pain; *C*, temperature.

In not a few cases of extramedullary compression, there is a striking difference between the degree of involvement of pain and of temperature sensibility. Pain is often profoundly affected at the same time that the ability to recognize and distinguish between different degrees of temperature is but little diminished (Fig. 7). *Per contra*, the power to feel the pain of a pinprick is sometimes well preserved in the same areas in

which hot and cold is not recognized as such, although extremes of temperature are felt as painful sensations.

It is difficult to find a satisfactory explanation for the dissociation of sensibilities that are supposed to pursue a common pathway, although many theories could be proposed.

The dissociation between tactile, pain and temperature sensibility was often marked in patients with extramedullary cervical cord tumors when the growth was located on the ventral or ventrolateral aspect of the cord (Figs. 5 to 8), and was distinct in several patients with com-

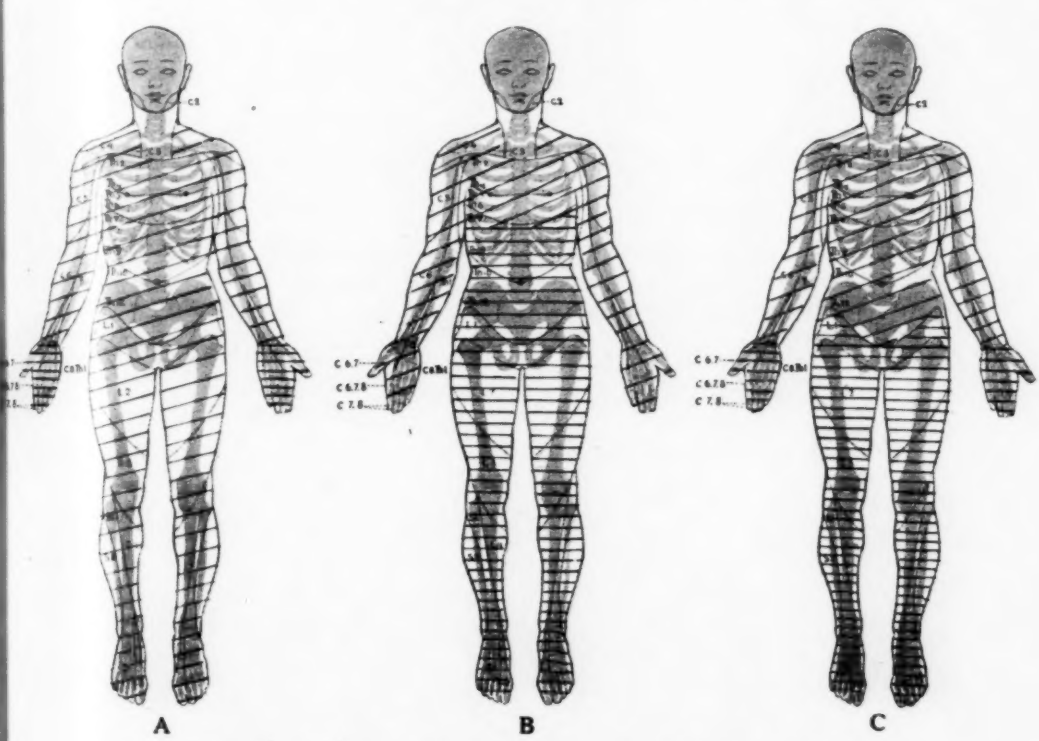

Fig. 8.—Cutaneous sensory disturbances in a patient (E. H.) with an extramedullary tumor on the right lateral aspect of the fourth and fifth cervical segments. Tactile sensibility only slightly diminished; *A*, tactile; *B*, pain; *C*, temperature.

pression of the cord by an extradural chondroma which had grown from the posterior surface of the body of a vertebra or the intervertebral cartilage (Fig. 9).

It has been a prominent feature in almost all of these patients with little or no disturbance of tactile sensibility and marked diminution of pain and temperature sensibility, especially if the growth lay on the ventral aspect of the cord, that the motor disturbances were wholly out

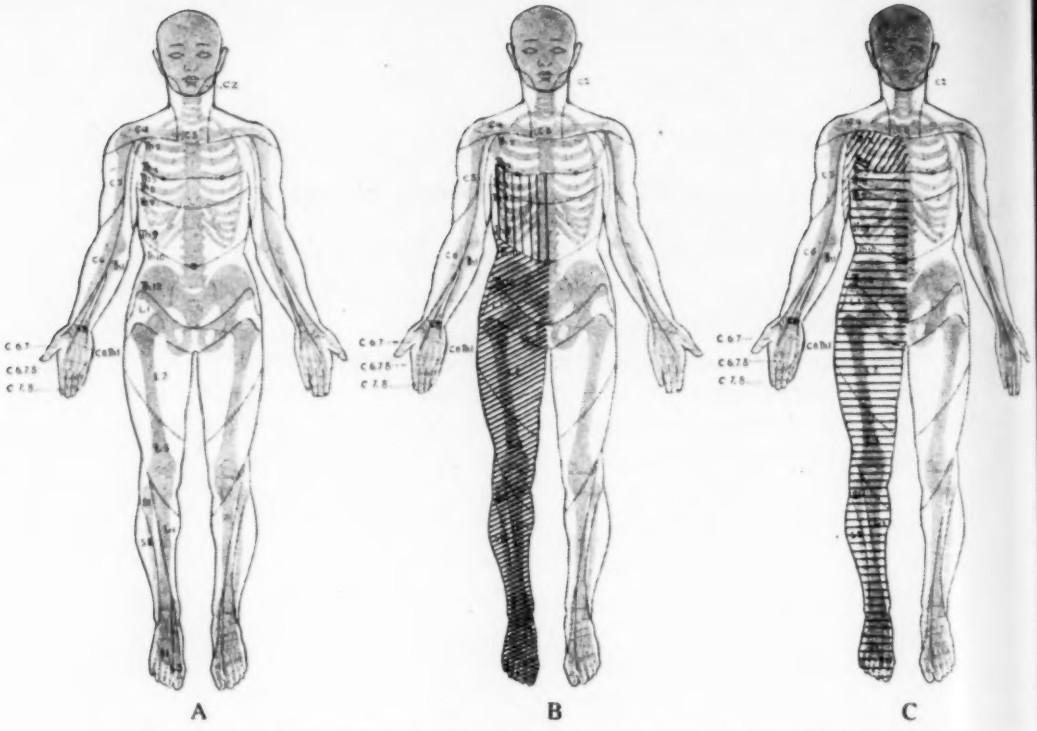

Fig. 9.—Cutaneous sensory disturbances in a patient (R. R.) with an extradural chondroma ventral to the fifth cervical segment. Tactile sensibility normal; pain and temperature involved only on one side; *A*, tactile; *B*, pain; *C*, temperature.

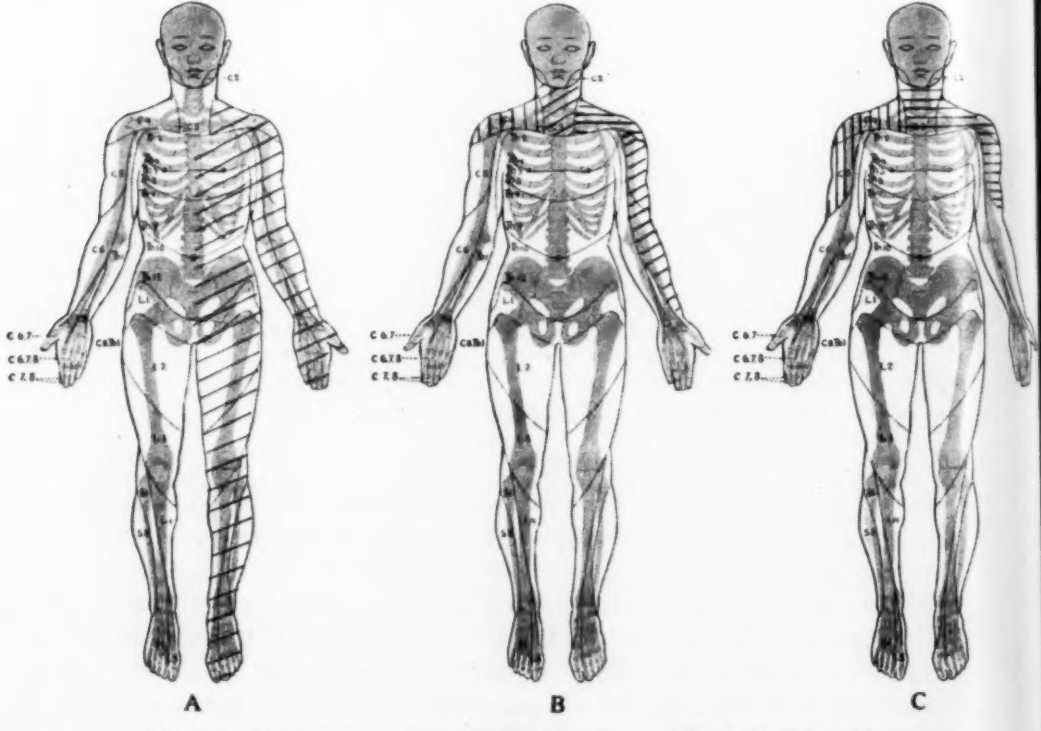

Fig. 10.—Cutaneous sensory disturbances in a patient (J. R.) with extramedullary compression of the upper three cervical segments from the left with marked motor but relatively few sensory disturbances; *A*, tactile; *B*, pain; *C*, temperature.

Fig. 11.—Spinal tumor in a patient (J. R.) as it appeared at operation.

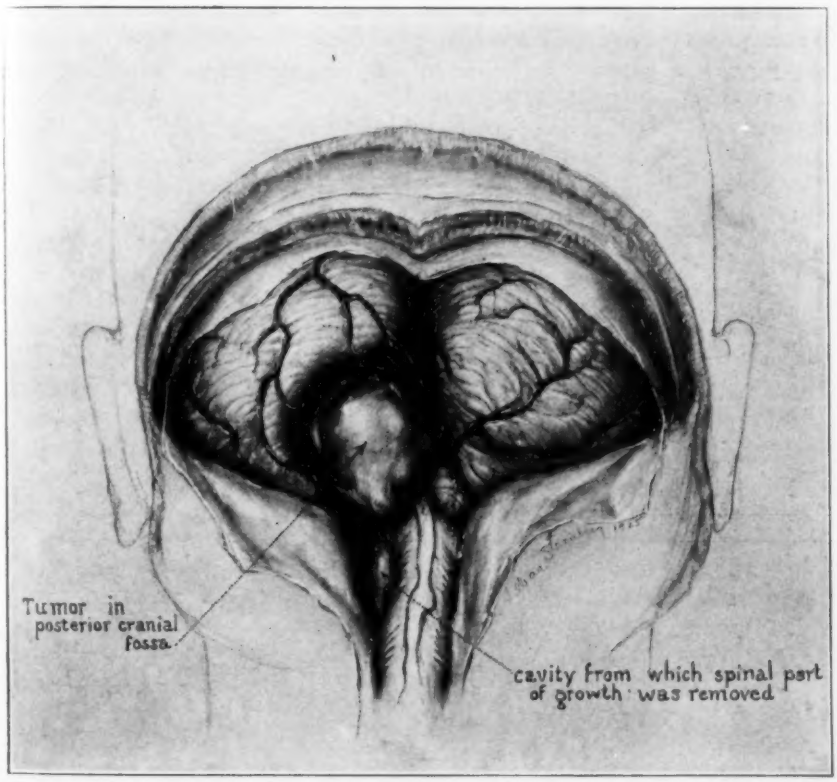

Fig. 12.—Tumor in the posterior cranial fossa found at postmortem examination.

of proportion to the sensory disturbances. Recently one of us has operated on a patient with signs of a marked compression of the first, second and third cervical segments by the downgrowth into the spinal canal of a posterior fossa endothelioma. In this patient the loss of power in all four limbs was advanced, although the sensibility disturbances were exceedingly limited (Figs. 10, 11 and 12).

CONCLUSIONS

1. Dissociation between tactile and pain and temperature sensibility is so frequent in extramedullary tumors of the cervical cord that this dissociation can be used only with circumspection for the distinction of intramedullary from extramedullary disease.

2. The greater involvement of the more caudal dermatomes in extramedullary compression and the more marked sensory alteration near the segmentary level in intramedullary growths is more reliable evidence for the differentiation between extrinsic and intrinsic cord disease due to tumor.

3. Unequal involvement of pain and temperature sensibility is frequently observed in compression of the cervical cord, in which the motor disturbances are often out of proportion to the sensory disturbances.

4. Many of the sensory disturbances observed in extramedullary and intramedullary compression are valid evidence in support of the belief that there is a lamellar arrangement of pain and temperature fibers in the lateral spinothalamic pathways.

GROSS PONTILE BLEEDING IN TRAUMATIC AND NONTRAUMATIC CEREBRAL LESIONS*

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AND
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The discovery of a pontile hemorrhage in addition to subdural and cerebral hemorrhages in a case of head trauma, prompted us to investigate the pons in other cases of this sort coming to necropsy. We also paid particular attention to the pons in cerebral vascular disease, abscess and tumor, and were surprised to find that pontile bleeding is not uncommon in them and in traumatic lesions. The importance of pontile hemorrhage, especially in traumatic cases, can be readily appreciated because it vitiates, in part, signs and symptoms which ordinarily might be of significant localizing value and might thus lead to operation. Conjugate deviation of the eyes and head, often considered a cerebral sign, might be produced by the pontile lesion and so give rise to an erroneous localization diagnosis. The frequent absence of cranial nerve paralysis in pontile hemorrhage may be misleading, and is explained by the fact that the hemorrhage usually occurs in the upper half of the pons, which is devoid of cranial nerve nuclei.

In view of the fact that our paper is concerned with hemorrhage in the pons, it is appropriate that at this point a brief description of its arterial supply be given. Stopford[1] says "the pontine branches of the basilar artery may be divided according to course and disposition into two sets: (*a*) A median set composed of minute branches arising from that surface of the basilar lying in contact with the pons, which at once enters the substance of the brain along the median groove. Duret subdivided this set into three groups, but no advantage can be gained by this; nor did my own observations justify it. Certainly they are more numerous caudally, where many enter the sulcus between the pons and bulb together with branches from the vertebrals, and again at the cephalic extremity of the basilar; but between these points they are generally found continuously with no suggestion of any definite

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1. Stopford: J. Anat. & Physiol. **50**:130, 255; **51**:250.

grouping. (*b*) A set of traverse rami which extend laterally and subdivide as though preceding into smaller branches which penetrate the ventral surface of the pons at right angles to the parent vessel. These vessels were generally arranged symmetrically on the two sides but were variable in size and number. . . . Obviously the various pontine branches of the basilar must, practically alone, supply under normal conditions the whole of the pons. . . . The median set supply the structures immediately lateral to the middle line in the tegmental part and the greater proportion of the pyramidal bundles and transverse pontine fibers in the basilar part as well as the more medial part of the corpus trapezoidum. The perforating branches from the transverse set supply the more lateral parts of the pons."

An insignificant source of supply to the pons consists of small branches from the anterior inferior cerebellar artery to the caudal part of the pons. From the superior cerebellar artery a few irregular branches penetrate the upper pons as the artery extends laterad from its origin. A few branches are given from the internal auditory artery.

The following records of cases, for some of which we are indebted to Drs. Burr, Potts, Solis Cohen, Gilpin, Spiller and Riesman, are illustrative of pontile hemorrhage occurring in a variety of pathologic conditions.

REPORT OF CASES

CASE 1.—*History.*—A colored man was brought to the psychopathic ward of the Philadelphia General Hospital, June 10, 1924, by a friend who knew nothing about him other than that the patient was acting queerly.

Examination.—The patient was confused, answered questions slowly and inaccurately, and was disoriented as to time and place. He had neither delusions nor hallucinations, and had no insight into his condition. Part of the mental state was thought to be due to word deafness. The head showed no evidence of injury. The pupils were slightly irregular, equal and reacted sluggishly to light. No paralysis could be made out, but weakness of the entire right side and of the left upper extremity was present. The patient was unable to perform coordinated movements, and a marked degree of apraxia was present. The knee jerks were equally exaggerated. Ankle clonus was not obtained, and plantar stimulation produced flexion on both sides.

Spinal puncture at this time revealed a fluid normal in every respect with the exception of a slight increase in the pressure. All laboratory tests were negative.

Clinical Course and Outcome.—On the fifth day after admission, alternating periods of tachycardia and bradycardia developed and the patient became comatose. The pupils were unequal, the right being the larger, and neither reacted to light. The eyes were fixed straight ahead. Both lower extremities and the left upper extremity were spastic; the hypertonicity varied at intervals of about one minute when waves of movement occurred which resembled the actions of a decerebrate animal; this was especially noticeable in the lower limbs, but also occurred in the left upper extremity; the right upper extremity remained flaccid. Babinski's sign and reflexes of defense were present on both sides. The head was turned

to the left, the neck and jaws were rigid and saliva ran from the mouth. The upper abdominal reflexes were lost. The eye grounds were normal, and a roentgenogram revealed no evidence of a cranial fracture. The temperature rose to 101 F. before death on the sixth day.

Pathologic Report.—The brain was of normal size. The left cerebral hemisphere was compressed by an overlying clot which was well organized, adherent to the dura and extended from the tip of the frontal lobe over the entire hemisphere. On the superior surface of the right hemisphere smaller organized subdural clots were present. When the brain was cut no area of hemorrhage was made out, but a transection of the pons above the level of the fifth nerve showed multiple hemorrhages, most marked on the right side, in both the base and the tegmentum.

Case 2.—*History.*—A white man, aged 54, admitted to the Oakbourne Epileptic Hospital and Colony Farm, July 15, 1924, had been an epileptic for the greater part of his life. While at the colony he had a great many spells, most of them of the major variety. All laboratory reports and the examination of his eyes were negative. Jan. 2, 1925, he had a severe fit and in falling struck and bruised his left mastoid region. He regained consciousness in a few minutes and apparently was all right, although he complained of headache for three weeks. February 4, he had another fit, struck the back of his head and sustained a slight cut. Later on this same day he had another fit, vomited, became unconscious and died in four hours. The temperature was normal throughout.

Pathologic Report.—Behind the left mastoid region an area of soft edema was present and a hematoma, not yet organized, 6 cm. in diameter, was discovered under the scalp. When the skull was opened free blood was found beneath the dura, especially pronounced on the right side. Several large subdural clots, apparently of recent origin, from 4 to 6 cm. in diameter, were spread over the right motor cortex and right frontal lobe, and smaller clots were seen on the left side near the base at the frontal pole. At the right frontal pole an area of dense adhesions between the dura and the brain was seen just behind the frontal sinus and a small amount of dark extradural pus was present in this location. A large hemorrhage, 4.5 cm. in depth and 3.5 cm. in width, was seen in the left occipital lobe, with an area of necrosis surrounding it. On section of the pons four large and many small hemorrhages were scattered through the cut section, mainly in the basis pontis; no hemorrhage was found below the level of exit of the fifth nerve.

Case 3.—*History.*—A white man, admitted to the psychopathic ward of the Philadelphia General Hospital, Jan. 17, 1924, in a confused state, while walking about the yard eleven days later was struck in the right malar region by a swing on which a boy was swinging. He was knocked to the ground, his head striking with considerable force. He resisted being placed on a stretcher and tried to get off several times. After he was taken into the ward he walked to his bed, but it was noticed that he dragged his right foot. He became quiet shortly after he was placed in bed. At this time a large swelling appeared over the right cheek.

Soon after admission to the ward he became stuporous, the respirations became infrequent and the pulse rate dropped from 80 to 60 within an hour. Further examination revealed that the pupils were fixed, unequal and irregular, the right being the larger. All the deep reflexes were increased, with ankle

clonus on the right and an abortive clonus on the left. Plantar stimulation produced flexion of the toes on the right and an uncertain response on the left. The abdominal reflexes were absent. Manipulation of the extremities failed to reveal paralysis in any part and marked resistance to handling was present. About three hours after the accident all the extremities became relaxed and the arms when passively elevated, dropped to the bed apparently paralyzed. The coma deepened and marked dyspnea and cyanosis developed. Four hours after the accident a lumbar puncture was performed and 15 c.c. of a bloody fluid withdrawn. After the puncture the patient became incontinent, the knee jerks

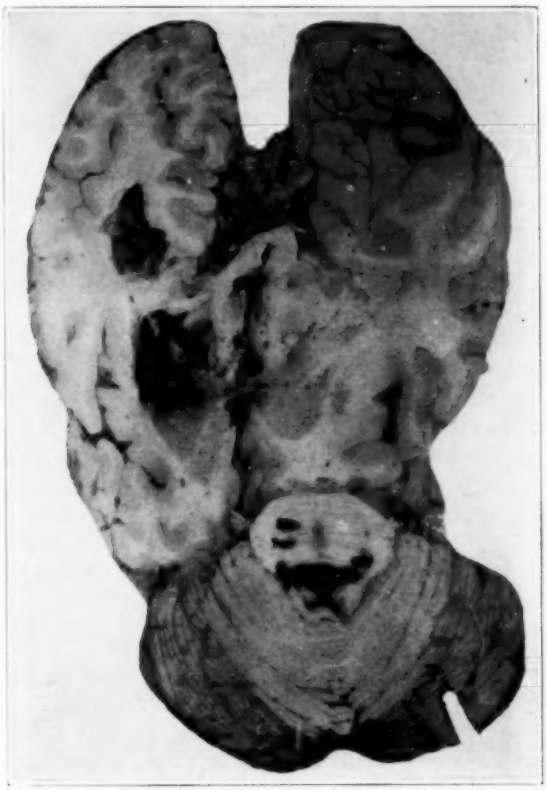

Fig. 1.—Multiple hemorrhages in left basal ganglia region and in pons.

were markedly diminished and Kernig's sign was present on both sides. Edema of the lungs occurred and the patient died, five and a half hours after the accident. The temperature was about 98 F. during the entire stay in the hospital.

Pathologic Report.—After the skull cap was removed the entire cerebrum was bathed in blood, but no fracture of the skull was found. An immense clot, not yet organized, was present in the right frontal region, compressing the right frontal lobe and extending to the rolandic area. A smaller clot was noted in the left frontal region. On cross section of the brain after hardening, an area

of recent hemorrhage was discovered in the left lenticulocapsular region, and two other areas were seen in the tips of the frontal lobes. The pons, both in the tegmentum and base, was the site of marked hemorrhage, but the medulla was normal (Fig. 1).

CASE 4.—*History.*—A white man, aged 75, who was admitted to the Philadelphia General Hospital, March 29, 1923, and died two days later, had been drinking and, several hours prior to admission, fell down a flight of steps. Shortly thereafter, he became stuporous but could be aroused.

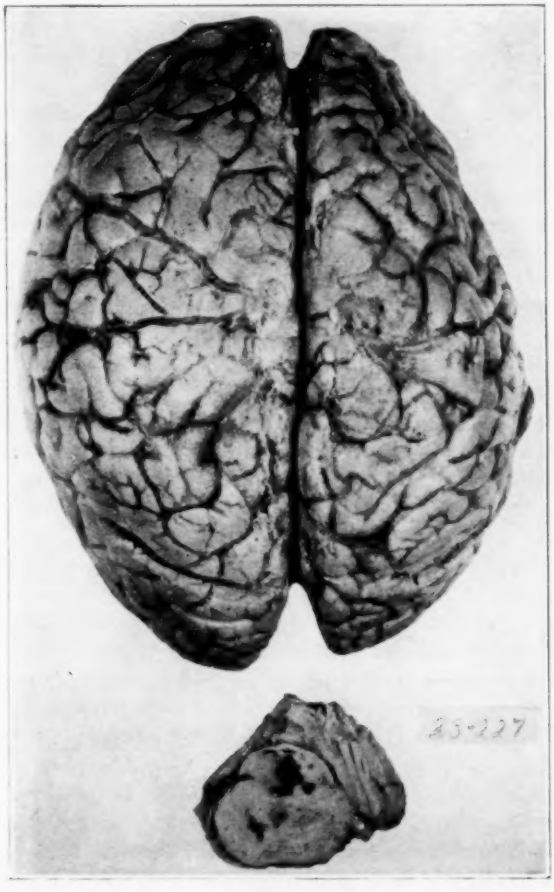

Fig. 2.—Right hemisphere smaller than left; pontile hemorrhages visible.

Examination.—There was a flaccid paralysis of the left side. The right pupil was larger than the left, and both reacted well to light and in convergence. Plantar stimulation on the left side produced extension of the great toe. He gradually became more and more stuporous, and died two days later. The temperature was never above 99 F. and most of the time was subnormal.

Pathologic Report.—The right hemisphere was larger than the left and was quite soft. The parietal area was the seat of a fluctuating mass which proved

on section to be hemorrhage and softening. Section through the pons above the level of the fifth nerve showed bilateral areas of hemorrhage in the tegmentum.

CASE 5.—*History.*—A white man was admitted to the Philadelphia General Hospital, Oct. 17, 1923, with a history of having been struck by an automobile eleven days before. He was taken to another hospital at which the scalp was repaired. The informant saw him after ten days when he talked rationally but complained of pain in the head and back. In the next few hours the patient became confused, did not know where he was, wandered about and could not sleep. The following day he became drowsy and could neither walk nor talk.

Examination.—The patient was elderly, comatose and breathing slowly and deeply. The pulse rate was slow and full. A partially healed wound was present in the midline at the parietofrontal junction. The pupils were equal, regular and very small, and did not react to light. The neck was moderately rigid and the abdomen scaphoid. The deep reflexes were universally increased and a bilateral ankle and patellar clonus were easily brought out. Plantar stimulation produced on both sides extension of the great toe. The temperature did not go above 99 F.

Lumbar puncture revealed straw colored and slightly cloudy fluid; the cell count was 8, and a few red blood cells were present; otherwise the fluid was normal.

The man died eight hours after admission to the hospital.

Pathologic Report.—A fracture of the right middle fossa 2 cm. in length was found. A brownish discolored area was discovered over the right temporoparietal region of the brain, and a similar but smaller area was present over the tip and under surface of the left temporal lobe. When the pons was sectioned in the upper part an extensive hemorrhage in the tegmentum and basis was disclosed (Fig. 2).

CASE 6.—*History.*—A white man, aged 39, was admitted to the Philadelphia General Hospital in a semi-comatose state after he had been drinking for one week. On admission, his temperature was 99.6 F. He had rigidity of the neck and bilateral Kernig and Babinski signs.

Spinal puncture was done and 10 c.c. of fluid removed; it was clear, under slightly increased pressure, and acellular.

Clinical Course and Outcome.—In the course of several days the man became sufficiently clear mentally to admit that he had been drinking. Four days after admission he was to have been discharged, but it was noted that he had a tendency to fall backward when standing. He was therefore kept in the hospital another day. He became worse, did not recognize his relatives and was confused and negativistic; he gradually became more stuporous and reverted to a condition similar to that on admission. The knee jerks were diminished and abdominal reflexes absent. The pupils were fixed and irregular. The spinal fluid was apparently under high pressure; 40 c.c. was withdrawn and showed nothing abnormal. The blood chemistry was: urea nitrogen, 29; uric acid, 6; sugar 190. He died seven days after admission, having continued in coma for the last three days.

Pathologic Report.—The brain was larger and softer than normal. The left temporal lobe was the seat of a necrotic tumor, indefinite in outline, 4 by 6 cm. in its greatest dimensions. It occupied the anterior part of the temporal lobe,

invading the inferior and middle temporal convolutions but not penetrating the superior. A hemorrhage, 1.5 cm. in diameter, was observed in the center of the tumor mass. It was noteworthy that the pons was particularly large, with small hemorrhages anterior to the aqueduct which was somewhat distorted as a result of pressure by the tumor. Microscopic sections showed the tumor to be a glioma.

CASE 7.—*History.*—A white man, aged 30, with unimportant past and family history, who was taken ill acutely Nov. 28, 1922, had had a tooth extracted because of an abscess several days before; following the operation occipital headache developed, and the right side of his body became numb. November 28, he became semi-stuporous; his speech was thick and he was paralyzed on the right side. He vomited for several days after the onset and had retention of urine for twenty-four hours. His wife said that he had complained of headache for the previous four years.

Examination.—The patient was semistuporous. Complete paralysis of the right side was present with rather more involvement of the upper part of the face than commonly occurs. All deep reflexes on the right were greatly exaggerated and those on the left side were somewhat increased. Plantar stimulation produced extension of the great toe on both sides. Pain, touch, temperature and position senses were seriously impaired on the right side. Hemianopia could not be determined.

Study of the eye grounds by Dr. Baer revealed choked disks with 2 diopters of swelling. The roentgenogram of the skull and examinations of the blood, spinal fluid and urine revealed no abnormalities. The cerebrospinal fluid pressure was 8 mm. of mercury.

Clinical Course and Outcome.—The man's condition improved somewhat and there was a slight return of power in the right leg. On Jan. 11, 1923, Dr. Charles H. Frazier performed an exploratory craniotomy on the right side. The dura was moderately tense and, in view of the possibility of an abscess, it was punctured, and an exploratory needle was introduced just behind the motor area, with negative results. On reflection of the dural flap an excessive amount of fluid escaped and this seemed to restore the subdural tension to normal.

Following the operation the man improved considerably and was discharged Jan. 31, 1923. He regained a moderate amount of power in the right lower, but very little in the right upper extremity. He continued to complain of headache and was irritable. He became suddenly unconscious Sept. 28, 1924, and died three days later. A record of the temperature was not available.

Pathologic Report.—The brain was large and a subpial hemorrhage was visible over the entire left rolandic area, especially in the midrolandic region, penetrating deeply into the cortex. Along the posterior margin of the motor area the cerebral tissue resembled a broken down gliomatous mass. On horizontal section of the cerebral hemisphere a clot 6 cm. in diameter and of the consistency of hard rubber was found extending from the periphery to the lateral ventricle on the left side, and destroying the basal ganglia and the motor and sensory areas. Section of the pons and medulla showed multiple hemorrhages above the level of the fifth nerve, particularly marked in the tegmentum and on the left side. The largest pontile hemorrhage measured 0.5 cm. in diameter. Microscopic examination of the tumor showed it to be a glioma.

Case 8.—*History.*—A white man, aged 50, who was admitted to the Philadelphia General Hospital March 1, 1925, gave an entirely inaccurate history of his illness; later, his brother gave the following account. About six weeks previously the patient began to act queerly while working at a greenhouse. His employer told him he should quit work for a while because of his bad memory. He then remained about his own home for a month, and during these weeks he spent much time walking about the house putting pieces of paper in flower pots as if he were setting plants. While his speech did not become affected so far as his ability to enunciate was concerned, his conversation became more and more unintelligible.

Examination.—The man was lying in bed apparently asleep, but he could be aroused. A marked kyphosis was present in the middorsal region, probably the result of old Pott's disease. Tenderness was present on percussing the right frontal area. The pupils and cranial nerves were normal except that there was a double choked disk. Station, gait, sensation and coordination were normal. All the deep reflexes were overactive and the right patellar was more increased than the left. All of the abdominal reflexes except the upper left were absent. The right cremasteric reflex was absent. Plantar stimulation on the right produced extension of the great toe.

The man was entirely disoriented as to time and place. His replies to questions were greatly delayed and he was unable to comprehend the meaning of many simple questions. When asked how old he was he replied, "38"; and when asked what was the year he gave the same answer. Sometimes he would respond to a question after it had been repeated a number of times. Ordinarily he took no interest in what was going on about him; at other times he was more alert although he never talked spontaneously. Apraxia was not present. Memory for recent and remote events was distorted; the patient stated that he was kicked by a horse three weeks ago when, as a matter of fact, this had occurred ten years previously. Tremor of the hands was noted, and the handwriting suggested general paralysis. The temperature did not go above 100 F.

Clinical Course and Outcome.—The patient remained in a state of mental hebetude from which he could be aroused. He was not incontinent, but made no effort to take care of himself. The tremor present in the right upper extremity became more pronounced. He spent a great deal of time fussing with the bed clothes and had to be fed; the food semed to collect in the right side of his mouth and about twice a day a large collection of food debris was removed. He had a series of epileptiform convulsions and died during one of them, four weeks after admission. The temperature ranged from 97 to 100 F.

Laboratory Examinations.—The roentgenogram of the skull and examination of the blood and of the urine revealed nothing abnormal. Lumbar puncture was not done.

Pathologic Report.—A large glioma was present in both frontal lobes, more marked in the right, leaving a comparatively narrow margin of the peripheral lobes intact. It was not sharply outlined, wholly destroyed the corpus callosum and completely obliterated the lateral ventricle.

On section of the pons at the level of the fifth nerves a hemorrhage, 2 mm. in diameter, was seen in the tegmentum; it became larger as the pons was ascended and extended into the midbrain. A smaller hemorrhage was found in the basis. Both of these areas of bleeding were in the midline.

CASE 9.—*History.*—A colored woman, aged 60, who was admitted to the Philadelphia General Hospital Nov. 21, 1924, had lost the power of speech about thirty-six hours before, had also difficulty in moving her tongue and developed a weakness of the right side.

Examination.—The patient was obese, with a blood pressure of 235 systolic and 114 diastolic. Her pupils were equal and regular and reacted well. Weakness was present on the right side with increased reflexes and ankle clonus, but without Babinski's sign. The lower left face was also weak; she could not move the tongue or soft palate at all, and had difficulty in swallowing. The urine showed a heavy trace of albumin, and hyaline and granular casts; the blood Wassermann reaction was negative to all antigens; the blood chemistry

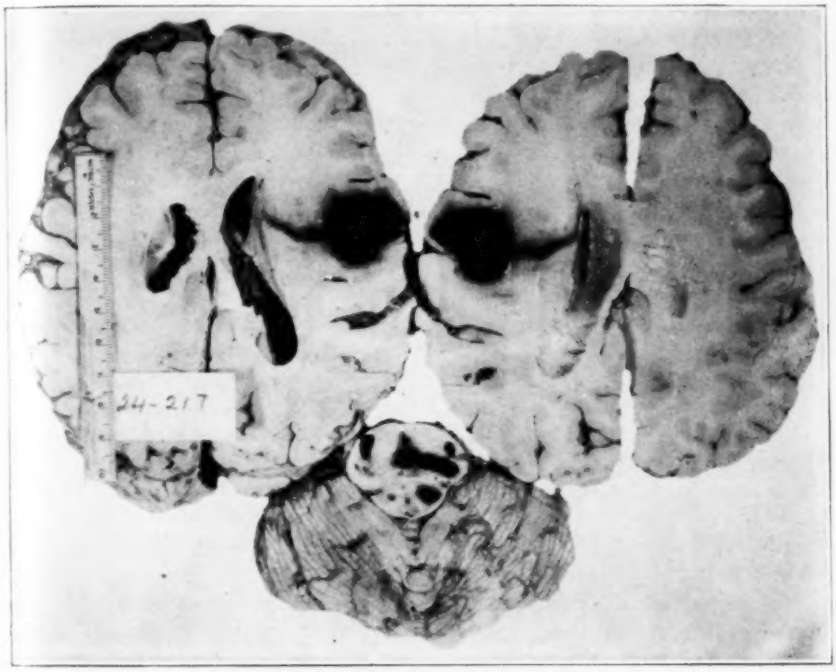

Fig. 3.—Hemorrhages visible in lower right motor area and in pons.

reports were: sugar, 150; urea, 45; uric acid, 6.8. The spinal fluid was normal. The temperature was between 99 and 100 F. until the day of death when it rose to 104 F.

She developed trophic sores on both buttocks, and died ten days after the onset of illness.

Pathologic Report.—The brain was small with tortuous, sclerotic vessels. On section of the cerebral hemispheres a number of recent hemorrhages were found; the largest, 3 cm. in diameter, was situated in the lower right motor area. Areas of hemorrhage were found in the caudate nuclei, in the right lenticulocapsular region, in the left occipital lobe and in the basis pontis (Fig. 3).

CASE 10.—*History.*—A white woman, aged 60, was admitted to the Philadelphia General Hospital Dec. 18, 1922, whose relatives stated that she had left home in her usual health that day and they had been notified at 7 p. m. that she was in the hospital. She had been picked up by the police in a greatly confused state. It was stated that she had had two previous "strokes" on the left side (the last three years previously), was able to walk, but could not use her left upper extremity. She was left handed.

Examination.—The patient lay quietly in bed and took but little interest in her surroundings. Her speech was thick, her answers irrelevant, and she was confused. Definite weakness of the entire left side and a probable left homonymous hemianopia were present. Her pupils showed nothing of importance. The blood pressure was 225 systolic and 130 diastolic. The reflexes on the left side were much exaggerated, with a Babinski sign but without ankle clonus. Because of her mental condition, sensation could not be accurately tested, although there was apparently loss of pain sense over the entire left side. The condition of the urine and the blood chemistry pointed toward a high degree of kidney disease. The blood Wassermann reaction was negative. The temperature ranged between 96 and 98 F. The patient died Jan. 5, 1923.

Pathologic Report.—A hemorrhage, 5 by 2 cm., was seen in the right lenticulothalamic region, with numerous punctiform hemorrhages surrounding the large one. A hemorrhage 2 cm. in diameter was noted in the pons close to the median line anterior to the right median fillet, just above the level of the exit of the fifth nerve.

CASE 11.—*History.*—A white man, aged 67, was admitted to the Philadelphia General Hospital Dec. 3, 1920, and died Oct. 7, 1921. On Nov. 23, 1917, he had been found unconscious, and when he regained consciousness had paralysis of the right side of the body and a speech defect. A similar, though much lighter attack occurred the following month. In April, 1918, he had a third stroke, which produced temporary weakness of the left side. When he was admitted to the hospital, motor aphasia, deviation of the tongue to the right and paresis of the right side of the body were present. He continued in this condition until three days prior to his death when he suddenly developed coma with rigidity of the musculature and hyperextension of his head.

Pathologic Report.—The brain showed very mild arteriosclerosis with convolutional atrophy, and the left side was larger than the right. An old softening was found in the left frontoparieto-occipital areas. A recent hemorrhage was present in the pons. The pineal gland was enlarged and cystic.

CASE 12.—*History.*—A white man, aged 49, with unimportant family and personal histories, was admitted to the Philadelphia General Hospital Feb. 17, 1925, complaining of weakness in the legs. This had come on gradually during the preceding seven months. The weakness increased so that at the time of admission to the hospital he was unable to walk.

Examination.—The ordinary signs of tabes dorsalis were present and, in addition, there was a tremor of parkinsonian type in the right upper extremity which was of one year's duration.

Clinical Course and Outcome.—Two days after admission the man became stuporous, and at that time an examination revealed that: the right pupil was twice as large as the left, the head and eyes were turned to the right, and there was slight left sided facial weakness and flaccidity of the left arm and leg. A

few hours later, the man vomited a number of times and died shortly afterward. The pulse rate and temperature were normal until a few hours before death when the latter rose to 101 F.

Laboratory Examinations.—The blood and urine were normal; the spinal fluid showed 3 cells to the cubic millimeter and a plus 4 Wassermann reaction to all antigens.

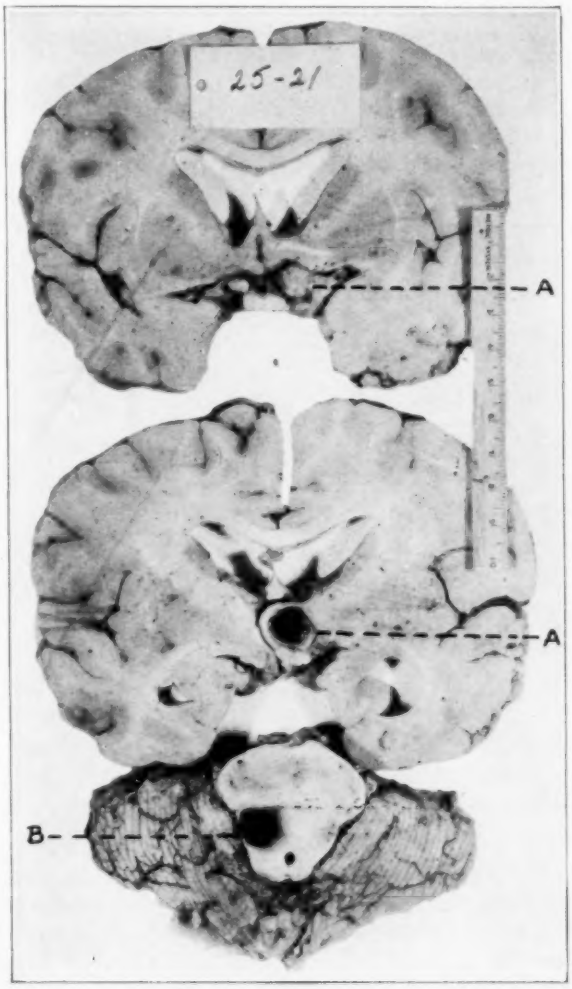

Fig. 4.—Frontal sections showing aneurysm, *A*, and pontile hemorrhage, *B*.

Pathologic Report.—The brain was large, with definite evidence of intracranial pressure; the blood vessels were small and sclerotic. An aneurysm, 1 cm. in diameter, of the first part of the middle cerebral artery was found which entirely filled the third ventricle. The aneurysm was filled with clotted blood but had not ruptured. A hemorrhage, 1 cm. in diameter, was found in the upper part of the pons, occupying the anterior portion of the left tegmentum (Fig. 4).

Case 13.—A white man, aged 36, was admitted to the University Hospital Nov. 10, 1915, complaining of general weakness. He had been well until a month previously. At that time a papilledema with hemorrhage was found in each eye. The most striking thing about this patient was the mental confusion; in speaking he would leave sentences unfinished and he had difficulty in finding the proper words. He was well developed and showed no evidence of visceral disease. The reflexes were a little more active on the right; but clonus and Babinski's sign were absent. Sensory examination was not satisfactory.

Clinical Course and Outcome.—There was a gradual increase in the choking of the disks but hemianopia did not develop. The man could not perceive odors through the left nostril, but was able to do so through the right. Nine days after admission he became unconscious but soon recovered. Five hours later he fell out of bed and could not be aroused. Lumbar puncture done at

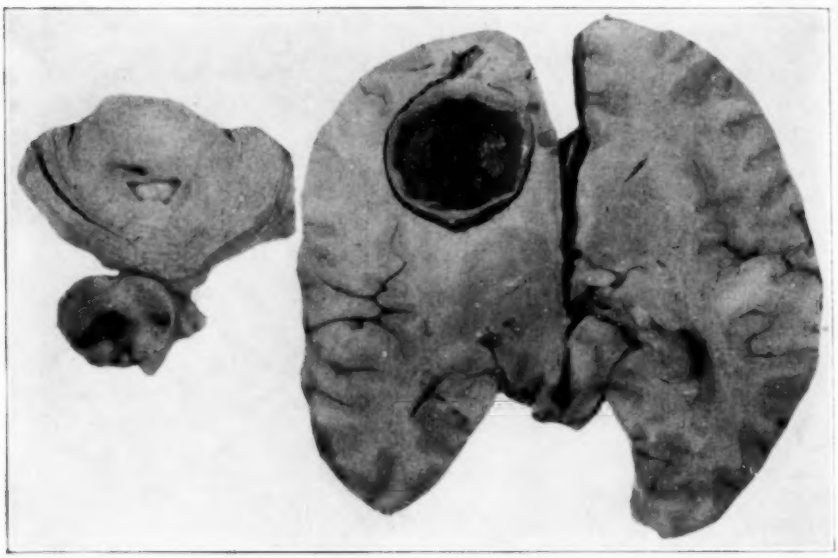

Fig. 5.—Frontal lobe abscess and pontile hemorrhages.

this time showed a pressure of 28 mm. of mercury and a clear fluid. The patient was transferred to the surgical service in a moribund condition and on the table his heart stopped. He was resuscitated and left the operating room in better condition than when he entered. One hour after the operation he suddenly developed symptoms of so-called medullary edema and died.

Laboratory Examinations.—The urine was normal and the blood showed nothing noteworthy except the presence of 13,000 white cells, of which 74 per cent. were polymorphonuclears. The temperature reached 102 F. on two occasions before death.

Pathologic Report.—An abscess was found in the left frontal lobe, approximately 5 cm. in diameter and with a wall 2 mm. in thickness. In the lower midbrain and in the pons several small hemorrhages were seen (Fig. 5).

COMMENTS

We have here reported the records of thirteen cases, each of which presented pontile hemorrhage, usually multiple; in addition, every brain showed an extrapontile lesion. Of the thirteen cases, five were traumatic, four vascular, three neoplastic and one an abscess.

The time of occurrence of the hemorrhage in the pons in the traumautic subjects is uncertain; one patient died five hours after receipt of an injury and another lived three weeks. It is also unknown whether the patients who survived more than a few hours developed the hemorrhage immediately after the trauma or whether it occurred some days later, although it is unlikely that they could long survive such marked pontile bleeding as they exhibited. In the first case that came under our observation, the man was confused and disoriented, but was not paralyzed; a few days later he became suddenly worse and developed signs and symptoms that were undoubtedly due to bleeding in the pons.

In Case 5, eleven days elapsed between the injury and the occurrence of pontile symptoms. In Cases 3 and 4, trauma of the head was followed in a few hours by signs of pontile involvement. In Case 2, injuries to the head were incurred both three weeks and four hours before death.

The time of occurrence of the pontile hemorrhage in the remaining cases could not be definitely determined, but it may have occurred shortly before and probably was the cause of death. The presence of bleeding in the pons in the tumor cases suggests the possibility that it may be something of this nature that causes the so-called medullary edema which is supposed to be a frequent cause of death in cerebral neoplasm as in Case 13.

The mechanism of the production of the pontile bleeding may be similar to that which causes a sixth nerve palsy in cases with increased intracranial pressure. Increase of intracranial tension causes pressure of the pons against the base of the skull and in this manner the sixth nerve becomes implicated. Pressure might likewise cause disturbance in the small vessels penetrating the pons from the posterior and lateral surface of the basilar artery. In the traumatic cases it is probable that the upper pons transmits the impact from one hemisphere to the other, and thus may be injured indirectly.

An interesting phase presented by these cases is the absence of noteworthy elevation of temperature except in one instance when it reached 104 F.

This series of cases of bleeding into the pons is taken from 129 necropsies of which twenty-six were traumatic, forty vascular, fifty-two tumors and eleven abscesses, making the incidence about 10 per cent.

Attwater,[2] in his paper on pontile hemorrhages, reported sixty-seven cases, with necropsy, which had occurred in Guy's Hospital during the preceding thirty-six years. This is by far the most comprehensive article in the literature on the subject of pontile bleeding; Greenacre[3] has also written on it, chiefly in its relationship to trauma.

CONCLUSIONS

Pontile hemorrhage occurs in trauma to the head, cerebral vascular disease, cerebral neoplasms and cerebral abscesses, ranging in frequency from 20 per cent. in the traumatic cases to about 6 per cent. in the tumors; this fact must be kept in mind in analyzing the symptoms in any acute cerebral condition. A strong possibility exists that at least some cases of so-called medullary edema are the result of pontile hemorrhage.

DISCUSSION

Dr. Adolf Meyer, Baltimore: I should like to refer to an investigation that was published in 1916 in the *Bulletin of the Johns Hopkins Hospital* and undertaken by Dr. Willis Greenacre, in the service of Dr. Le Conte, under the direction of Dr. Bassoe, with very similar results, which shows clearly the necessity of our bearing in mind multiple lesions, and particularly multiple lesions of this sort.

In the early days of lumbar puncture, when fatal accidents occurred, we found forward dislocation of the pons causing obstruction with multiple hemorrhages; this suggests the possibility that there may be in various lesions, and especially in hemorrhages in one part of the brain, kindred dislocations and effects on the circulation that favor the occurrence of hemorrhages in the dislocated region. Hardening of the brain in situ might give us light on this possibility.

Dr. William G. Spiller, Philadelphia: I take it that Dr. Wilson and Dr. Winkelman did not mean so much to imply that multiple lesions were common, as to call attention to the fact that especially is the hemorrhage likely to be in the pons, as distinct from other parts of the brain. The explanation in most cases is to be found in the fact that those conditions which lead to a hemorrhage in the cerebrum are the same as, or similar to, the conditions which lead to the hemorrhage in the pons.

Not only is it true that hemorrhage in the cerebrum is likely to be associated with hemorrhage in the pons but so also is softening from occlusion of vessels.

I have been struck by the frequent finding, over a long period of years in specimens in my laboratory, of thrombotic lesions in the center of the pons, when I have found thrombotic lesions in the cerebrum, and have explained it in the way that I have just mentioned.

Dr. Hermon C. Gordinier, Troy, N. Y.: The authors reported one case in which the temperature rose as high as 104 F. In hemorrhage of the pons that temperature is not at all unusual. I therefore ask whether high temperatures were observed in cases of multiple hemorrhages in the pons.

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2. Attwater: Guy's Hospital Reports **65:** (**50** of the third series) 339, 1911.
3. Greenacre, P.: Bull. John Hopkins Hosp. **28:**86 (Feb.) 1917.

Dr. Charles K. Mills, Philadelphia: This paper is of interest to me in connection with a case reported by me many years ago. The paper reporting this case was entitled, "Hemorrhage into the Basal Ganglia Followed by Effusion of Blood into and Beyond the Ventricles, Symptoms and Lesions of Apoplectic Shock, Cheyne-Stokes Respiration, High Head-Temperature, Etc.," *The Philadelphia Medical Times* **11**:41-44 (Oct. 23) 1880. It was a case of large hemorrhage at the usual seat of cerebral hemorrhage, the hemorrhage having broken eventually into the ventricles. There were, besides, multiple hemorrhages all over the surface of the brain in the membranes and minute hemorrhagic extravasations in the pons and floor of the fourth ventricle.

The hemorrhages, I believe, are due, as Duré pointed out in his cases of traumatism in his dog experiments, to the sudden universal displacement of cerebrospinal fluid in such a way that immediate conditions of vacuolation are created at many points around the small vessels. The external support and pressure being removed, hemorrhages take place in consequence into the vacant spaces. A sudden hemorrhage into the interior of the brain strikes the brain a blow from within, which is comparable to the blow on the cranium from without. The blows from within and from without produce similar disturbances in the brain.

Dr. E. D. Friedman, New York: I observed a young man who was struck a blow between the eyes. Following that he developed stupor, low-grade papilledema and evidence of bleeding into the subarachnoid space. After a few days, he complained of diplopia which on further investigation was found to be due to a divergence paralysis. In conjunction with this there was a moderate ptosis of the upper lids and a little difficulty in upward gaze. We interpreted that at the time as an evidence of bleeding into the brain stem probably in the periaqueductal region.

Dr. Michael Osnato, New York: Was there anything in the pontile vessels to explain the delayed hemorrhage in the case in which death occurred after eleven days?

Dr. Joseph H. Globus, New York: In a study of a fairly large number of cases of cerebral hemorrhage associated with pontile bleeding, we find that the hemorrhages occur in preexisting cavities. No matter how suddenly extravasations occur, the cavity or cavities are usually found lined by a wall or a substance which gives evidence of the preexistence of the cavity.

In a number of instances we have found cystic areas, varying in size, in the pons, not containing blood. It is quite possible that under other conditions the cystic cavities could have been filled with blood, and thus, instances similar to those presented by Dr. Winkelman and Dr. Wilson obtained.

It is likely, that the very factor which produces a hemorrhage in the cerebrum may produce a similar hemorrhage in the pons, provided the pons contains features which prepare it for such an effect. If you have in the pons a small cystic cavity or a small area of encephalomalacia, the same force which produced a large area in the cerebrum might have produced an area in the pons.

Dr. Hugo Mella, Denver: Thinking of the mechanics of this type of lesion, I thought of the return circulation from this area. I believe that most of the return circulation is through vessels tributary to the vein of Galen. Isn't it true that most of our traumatic cases and also in our cerebral hemorrhage cases there is a certain amount of edema, and wouldn't it be reasonable

to expect that there is a disturbance of the return circulation through the vein of Galen? That is, that there is a compression of that vein, a backing up of blood in that region, and possibly that is why we find hemorrhage in regions drained by it.

DR. N. W. WINKELMAN: The paper referred to by Dr. Meyer had escaped our notice.

Dr. Spiller answered Dr. Meyer's question. We did take softening in the pons into consideration, but we felt this paper was long enough without such discussion.

We looked very carefully for hyperthermia and found it only in one out of the thirteen cases of pontile lesion.

We read Dr. Mills' paper and our own theory was modeled in part on this.

The description of the case that showed bleeding around the aqueduct is very similar to one that we showed on the screen, in which the hemorrhage did extend along the aqueduct, in the midbrain and down into the pons, and I can see very readily how the symptoms could be produced.

THE CORRECTION OF COLLOIDAL GOLD SOLUTIONS AS APPLIED TO THE LANGE REACTION

A SIMPLE AND ACCURATE TITRATION METHOD

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It has been the general experience in the clinical laboratory that the preparation of a proper colloidal gold solution, that is, a solution which will prove ideal for carrying out the colloidal gold test as applied to the differentiation of cerebrospinal syphilis, is often fraught with certain difficulties frequently not under the control of the worker. With due regard for the proper cleansing of glassware, the purity of the reagents entering into the preparation of such a solution and temperature precautions as laid down by those[1] who have investigated the nature of colloid chemistry as highly necessary prerequisites for the achievement of a good product, it is not infrequently found that such solutions thus prepared are far from being suitable, if not altogether unsuitable, for the test proper.

Excluding those colloidal gold solutions that are grossly bad or self-condemnatory by their physical appearances, that is, either turbid or of a bluish color, there are in practice two kinds of solutions which are confessedly unsuitable for the reaction proper. One is the so-called highly "protective" solution, which is so fixed in its colloidal state that when added to a known pathologic cerebrospinal fluid, in its proper dilution in the test, it produces a flat number one reaction "curve" or, at best, a number three reaction curve in the very high dilutions after considerable long-standing. In other words, it will give an ideal negative curve for normal fluids and a false "tabetic" curve with a known "paretic" fluid. Another solution, while it meets in its physical appearances the requirements for a proper product, is found on preparation to be highly "sensitive" or loosely bound in its colloidal state, so that when subjected to test with a known pathologic spinal fluid, it will precipitate the first five serial tubes, and proceed to do so within thirty minutes in the higher dilutions, forming a flat number five reaction "curve." With a known normal fluid such solutions will form a false "tabetic" curve to start with, and within from twelve to fifteen hours will cause a precipitation in the lower dilutions, or the first tubes

1. Young, S. W.: Chapter on Colloids in Zinsser, Hans: Infection and Resistance, Ed. 2, New York, The Macmillan Company, 1918.

of the series, as well. In physical appearance it is indistinguishable from the so-called "protective" solution.

Since the colloidal gold reaction has been universally accepted as of great importance in differentiating tabes from paresis and other forms of syphilis, it is evident that such improper solutions are "dangerous." The general emphasis laid on the preparation of the colloidal gold solution as of greater importance than the test proper is wholly justified.

The method generally accepted as the best for obtaining a good solution is the reduction method of Miller and his co-workers.[2] By this method, triple distilled water is brought to a temperature of 60 C., at which point the reagents (1 per cent. gold solution, 2 per cent. potassium carbonate and 1 per cent. oxalic acid solution) made up in triple distilled water are added; it is finally brought up to 90 C., and with constant stirring a 1 per cent. formaldehyd solution is added and agitated until the proper color is produced. This method will be found in almost every standard textbook on serology. Behrens,[3] in a recent study of causes of unsatisfactory colloidal gold solutions, believes that the age of the potassium carbonate is invariably at fault, and advises that this reagent be used when not more than twenty-four hours old. The primary cause of failure to obtain a suitable solution is generally conceded to be the reaction of the final product. A solution must be absolutely neutral in order to be useful. Slight deviation from the point of neutrality, on either the alkaline or the acid side, will be found unsatisfactory. As a rule, a colloidal gold solution which is slightly alkaline will be found to be "protective," while one which is slightly acid proves to be "sensitive." As pointed out, this unfavorable reaction, as primarily responsible for bad solutions, is unavoidable, and a method for adjusting or correcting such solutions is preeminently called for.

The method of titrating a definite quantity of the solution, using alizarin as an indicator and correcting it with fiftieth normal acid or alkali, as the case may be, is far from accurate, because of the fact that on the addition of the indicator to the already brightly colored solution, there are difficulties in determining the end point. Often one is unable to see any indication of the reaction; the indicator visibly does not indicate, as a matter of fact, in practice; this method of titration is rarely, if at all, utilized in the laboratory. Many a batch of colloidal gold solutions are discarded rather than corrected if they turn out to be unsatisfactory on preparation.

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2. Zinsser, Hans: Infection and Resistance, Ed. 2, New York, The Macmillan Company, 1918.

3. Behrens, C. F.: Nav. M. Bull. **21**:191-192.

The standard conditions which must be fulfilled by a good solution are laid out by Kolmer [4] and are beyond contradiction, indeed. One of these conditions is that 5 c.c. of a colloidal gold solution properly prepared must be able to precipitate itself in the presence of 1.7 c.c. of a 1 per cent. sodium chlorid solution within one hour's time. A solution of an absolute neutral reaction will respond to this preliminary test invariably, and will prove highly satisfactory when applied in the test proper. It will react negatively with a normal fluid and positively with a pathologic fluid.

Since sodium chlorid is an electrolyte and a capable precipitant of colloidal solutions, finding its application as an index of the state of dispersion of a given solution, it was thought that exactly 1.7 c.c. of a 1 per cent. solution of this salt, as used as a standard efficiency test and applied to colloidal gold solutions, would prove an ideal indicator in a titration method.

The method of procedure is outlined in the table.

Method of Procedure

Number of tube	1	2	3	4	5	6	7	8	9	10	11	Control
Twentieth normal sodium hydroxid or twentieth normal hydrochloric acid (as required), cubic centimeters	0.05	0.075	0.1	0.15	0.2	0.25	0.275	0.3	0.35	0.375	0.40	None
Colloidal gold solution (as prepared), cubic centimeters	5	5	5	5	5	5	5	5	5	5	5	5
1 per cent. sodium chlorid solution, cubic centimeters	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7	1.7

The series of tubes thus set up are set aside at room temperature, protected from light and read at the end of one hour. The tube showing complete precipitation and containing the least amount of acid or alkali is taken as the correction point for the solution. The amount needed for the whole is calculated and added to the prepared solution. The control tube serves as a preliminary indicator and shows whether acid or alkali is needed for correction. It is not advisable to use an acid or alkali solution of higher normality, because it would affect the strength of the sodium chlorid solution used as an indicator, an important factor.

It should be remembered that a colloidal gold solution at best, when tested with a clinically pathologic cerebrospinal fluid, does not precipitate in the first or second tubes, or high concentration, at once, but the higher dilutions (1:160 or 1:320) take precedence and precipitate

4. Craig, C. F.: The Wassermann Test, Ed. 2, St. Louis, C. V. Mosby Company, 1921.

almost immediately. This is probably due to the fact that the high concentration of a fluid that has a high protein content (globulins) brings about a condition of surface tension unfavorable for immediate precipitation. It should also be remembered that the method of titrations, as outlined, cannot be used for solutions grossly improper because of noncompliance with technical requirements.

CONCLUSIONS

1. In the preparation of a good colloidal gold solution, certain technical difficulties, at best unavoidable, are frequently encountered. Many solutions are discarded as unfit, though prepared with great care.

2. The primary cause of unsuitable solutions is the reaction of the final product.

3. The old method of titration, using alizarin as indicator, is not entirely satisfactory. It does not visibly "indicate" because of the primary color of the solution under titration.

4. A method of titration using 1 per cent. sodium chlorid solution to the extent of 1.7 c.c. as an indicator is suggested. This salt is an electrolyte and a precipitant of colloidal solutions, and serves as an accurate and highly satisfactory indicator of the reaction state of a prepared colloidal gold solution.

Clinical and Occasional Notes

DELAYED POST-TRAUMATIC INTRACRANIAL COMPRESSION

ABRAHAM O. WILENSKY, M.D.

NEW YORK

In neurologic surgery it is well known that craniocerebral injuries are often followed immediately by more or less pronounced conditions of increased intracranial pressure. It does not seem to be so well known that conditions of increased intracranial pressure causing alarming symptoms and requiring active therapeutic measures may make their appearance suddenly as a late phenomenon following craniocerebral injury, which need not be extensive, is often of negligible consequence, and occasionally passes unrecognized.

Such a case recently came under my observation.

REPORT OF A CASE

History.—The patient was a strong, active and robust man. Up to the time when he received his injuries, he was in the best of health, and there were no signs or symptoms of any disturbance or illness.

About six weeks before admission to Mount Sinai Hospital, the patient was involved in an automobile accident, during which he was thrown from the car and landed on the road some distance away. He sustained a fracture of the left femur in its upper third. Apparently there was no gross injury of the head. He was treated in a local hospital for the fractured thigh with a plaster of Paris cast for about two weeks. For three weeks more he apparently did well. Suddenly, the patient became irrational and delirious and rapidly thereafter he became drowsy. This unlooked for complication caused such consternation in the patient's family that he was immediately brought to the city by ambulance. During the transportation he received three or four hypodermic injections, each of one-half grain (0.03 gm.) of morphin, at hourly intervals.

This history when later amplified by close questioning of the patient and his family disclosed the significant fact that the absence of any injury of the head was only apparent. It was brought out that the man was dizzy, and not completely oriented for a short time after the injury, and that a moderate amount of headache persisted for several days. There was no external evidence of trauma, and the comparatively trivial character of the symptom caused the local attendant to neglect this part of the syndrome.

Examination.—On admission to the hospital the patient was in excellent physical condition. He was moderately drowsy and could be aroused fairly easily, but went back to his drowsy state as soon as he was left undisturbed. When aroused he showed a marked lack of memory associations, and was more or less completely disoriented. Because of these disturbances there was a complete lack of cooperation on the part of the patient in carrying on the examination.

There was no external evidence of any craniocerebral injury either visible or palpable. There was no rigidity of the neck and no Kernig's sign. There were no paralyses or rigidity of any of the limbs; the musculature was flaccid.

The eyes were normal externally. There were no ecchymoses. The pupils reacted to light and in accommodation. There were no external ocular palsies and no strabismus. The eyegrounds were normal, and there was no elevation of the disks. The nose, mouth and throat showed no abnormalities. The sense of smell could not be investigated. The ears showed no abnormalities to the ordinary forms of examination; tuning fork or other functional tests could not be made. The functions of all of the other cranial nerves apparently were undisturbed; there was no facial palsy.

There was no enlargement of the lymphatic glands. The thyroid gland was not enlarged.

The heart and lungs showed no abnormalities. The condition of the circulation was excellent. The pulse rate on admission was 120 to the minute. It fell by the second day to about 96, and thereafter it varied irregularly. The respirations varied between 28 and 36 to the minute.

Abdominal examination revealed nothing abnormal.

The line of the fracture of the left femur was easily palpable through the soft parts; the lower fragment was displaced upward. There was fair union between the fragments, and the fracture apparently was in an advanced state of healing.

The abdominal reflexes were not elicitable. There was a doubtful Babinski sign on the left side; there was no clonus; the knee reflexes were active and equal on the two sides. There were no other abnormalities of the reflexes.

Course of Illness.—At the time of admission the temperature was 103 F. By the next day it had fallen to 99 F. The general condition continued about the same and remained unchanged on the following two days. Examination of the patient did not furnish any new facts. The systolic blood pressure was 105 and the diastolic 75 mm. of mercury. The pulse rate was irregular and averaged about 90 beats to the minute. There were continual involuntary evacuations of the bladder and, occasionally, of the rectum. The urine itself contained no abnormal elements. A roentgenogram of the skull showed no fracture; that of the thigh showed the line of fracture at the junction of the upper and middle thirds of the bone, with considerable overriding and with much callus formation.

When the patient was first admitted, the physical signs and phenomena indicated that we were dealing with some form of intracranial compression, and we could quickly discard such possibilities as a meningitis, an encephalitis or a neoplastic formation. The first impression that we were dealing with a consequence of some form of injury of the head was corroborated later when we questioned the family carefully. Only then did the few facts appear which indicated that there had been an injury of the head of some kind, even though it was a minor one. However, at the time of the patient's first admission to the hospital the only course left open to us was to consider the case as one of intracranial compression, and to act accordingly.

On the fifth day after admission to the hospital the stupor had deepened and become marked; there was difficulty in arousing the patient. A lumbar puncture was performed the same evening; 70 c.c. of normal, clear cerebrospinal fluid was released from the subarachnoid space; the fluid was under considerable pressure, and escaped through the needle with great speed.

Following the lumbar puncture there seemed to be temporary relief in the symptoms of brain compression. The stupor lightened considerably. At this time the systolic blood pressure was 115. Within a few hours, however, the stupor began to deepen again, and quickly reached its previous condition. By

the afternoon of the sixth day the stupor was extreme. The general condition was still very good. The systolic blood pressure had meanwhile risen to 136 and the diastolic to 96.

Another lumbar puncture was performed the same evening. A large amount of clear cerebrospinal fluid was again released from the subarachnoid space with similar increased pressure and with equally great speed. During the next few hours the stupor lightened again, and after a few hours it again deepened to a depth even greater than had previously been present.

The cerebrospinal fluid showed few cells on microscopic examination; it contained no organisms, and cultures of the fluid remained sterile.

The first lumbar puncture was performed for two reasons: We wanted to substantiate our working diagnosis to be sure that no intracranial hemorrhage had taken place and to exclude an inflammatory condition such as a meningitis or encephalitis; and we wished to observe the therapeutic effect of the release of cerebrospinal fluid from the subarachnoid space. The diagnosis of intracranial compression was substantiated by the pressure of the escaping fluid and by its physical characteristics. The absence of old or new blood in the cerebrospinal fluid was only of negative value; only a positive response would have been of any clinical importance.

The improvement which followed the release of the cerebrospinal fluid, even though it was only temporary, gave true insight into the course of treatment which was to be followed. This encouraged us to make use of the method a second time. The rapid return of the symptoms following the temporary improvement which again followed the second lumbar puncture, indicated that the measure was only of a palliative nature and was unfortunately insufficient even on that basis. A more active and efficient method was apparently needed, and that, too, imperatively so, if regression of the rapidly advancing and increasing intracranial compression was to be accomplished. Some form of intracranial decompression was needed.

This experience with lumbar puncture corroborated those to which reference was made by me on another occasion. It has been my experience that lumbar puncture, as a general rule, is merely a palliative measure for the relief of intracranial pressure — and a poor one at that. Whenever states of increased pressure beyond the minor grades were present, a much more active and energetic measure was needed if any permanent good effect was to be expected; this invariably means a decompressive operation.

A right subtemporal decompression was performed late the same night under local anesthesia. A permanent opening measuring 3 by 5 cm. was made in the skull. On incising the dura a considerable amount of cerebrospinal fluid escaped into the wound. The brain was edematous and bulged somewhat into the wound. No other abnormalities were noted. The dura was left wide open, and the outer wounds in the muscles, fascia and skin were sutured in layers. No drainage was necessary.

The patient reacted very well from the operation, and almost immediate improvement was noticeable. The stupor began to lighten, and it seemed somewhat easier to arouse him. There was marked restlessness. The postoperative rise of temperature was small — 101 F. — and it subsided quickly.

There was progressive improvement thereafter, and a week after operation the stupor had entirely disappeared. The patient was noisy and restless, and was controlled with great difficulty. The bladder and bowel functioned perfectly.

At the end of the second week the patient began to have periods during which he was fully conscious and oriented, and when he remembered everything

that had happened prior to his coming to the hospital. These grew more frequent and lasted longer each time. During the intervals between these periods he was noisy, restless and abusive. The latter phenomena gradually disappeared.

Twenty-two days after operation the patient was discharged from the hospital at his own request. There was no evidence at the time of his discharge from the hospital to indicate intracranial disturbance. The wound in the head had healed completely by primary union. The fracture of the thigh had united solidly, and the patient was able to get about on crutches. Mentally he was not entirely oriented and in complete control of all of his mental faculties.

The follow-up history of this patient corroborated the permanence of the cure. I have seen this patient on a number of occasions since his discharge from the hospital, and physically and neurologically he has shown no deviation from the normal. The patient is able to get about in his daily work and shows no disability. His mental state is normal, and apparently there are no reliquae either from his intracranial compression or from his subtemporal operation. It is two years since the patient's discharge from the hospital.

There are numerous cases on record in which patients are admitted to a hospital with a craniocerebral injury in whom states of intracranial compression of greater or less severity make their appearance at periods of from seven to fourteen days after the injury. Such instances are, perhaps, but exaggerations of the common condition of increasing intracranial pressure after injury in which the maximum effects become visible and perceptible in a few hours. I speak of the sequence of events in which bleeding from the middle meningeal artery gives us the classic example. This instance in which the onset of the symptoms of intracranial compression occurred six weeks after the reception of the injury seems to be unique as regards the lateness of onset of the symptoms. Personally I have never had another such experience.

A condition has been described in which edema of the brain occurs suddenly and unexpectedly, and of which the clinical picture has certain resemblances to the one exhibited by this patient. This unexpected edema was described by Rawling, and in his experience this usually complicated insolation, or at least followed an undue exposure to extreme heat in the tropical countries. In the patient described in this report, there was no such exposure. The history of dizziness and disorientation indicated that some form of intracranial disturbance resulted from the accident which most probably was of the nature of a general brain contusion and which corresponded clinically with an intracranial concussion of rather mild extent.

Intracranial compressions of traumatic origin which occur at late periods most commonly must owe their existence to a slow bleeding within the cranial cavity or from and within the brain substance. It seems almost impossible to believe that such slow bleeding could continue for six weeks in the absence of any bleeding tendency. The exploration during the decompressive operation did not show any old or active bleeding in the immediate environment of the cranial wound. This, however, does not disclose the conditions elsewhere in the cranial cavity. The notes of the following case of Degee which I quote from the literature are interesting in this regard:

"A 54 year old hostler suffered a fall during which he received a contusion of the left frontal region and a contusion of the shoulder, and which was followed by unconsciousness but not by vomiting. There was a rapid recovery, and the patient was able to go home unaided. The pulse, temperature and respiration were normal. The superficial wound healed in ten days.

"Entirely without symptoms and without headache, the man was able to work for the next four weeks. During the eighth week, headaches began, and several days later, he suddenly developed a general disturbance of his mental faculties, and he complained of not feeling well. Finally, a left hemiplegia developed, then an aphasia and apraxia. The terminal stage was marked by convulsions and unconsciousness, and death followed eight weeks after the injury.

"Postmortem examination showed the dura over the left anterior lobe to be dark brown and pigmented. On incision of the dura a subdural cavity was exposed which contained old blood."

COMMENT

In my patient there was a considerable excess of cerebrospinal fluid above the normal as demonstrated in the lumbar punctures and at operation. Edema of the brain frequently occurs, with cranial and intracranial trauma, and is frequently sufficient to end fatally. It is possible that the essential nature of the lesion was such. In the absence of any further data, owing to the fortunate recovery of the patient, a definite opinion concerning the actual intracranial pathology is not possible.

Obituary

HENRY RUST STEDMAN, M.D.

1849—1926

After a period of failing health, which extended over several years, Dr. Stedman died on February 19, at his home in Brookline, Mass., under the devoted care of his younger daughter, who alone of his family was living with him at the time of his death. He was the fourth in his family to carry on the tradition of medicine; his brother, his father and his grandfather were also physicians. He was born in Boston, Sept. 19, 1849, received his preliminary education at the Latin School, and was graduated at Harvard College in 1871, and at the Medical School of Harvard University in 1875. He was further prepared for his subsequent professional work by a surgical service at the Massachusetts General Hospital and by a medical house-officership at the Boston City Hospital. His interest soon turned to the study of mental disease and, to perfect himself in this branch, inadequately taught at that time, he became assistant superintendent of the Danvers (Mass.) State Hospital for the Insane, and later still further prosecuted his chosen field of work by residence at the Edinburg Royal Asylum, and the West Riding Asylum at Yorkshire, England.

Soundly and broadly equipped by this long period of preliminary training, he returned to America, and, in 1884, established a small hospital at Forest Hills, later removing to Brookline, for the treatment of mental disease. This hospital from the first was the center of his medical interest, and throughout his active life he gave to it his most painstaking attention, with the result that it has always stood and still stands, under the direction of his successor, Dr. George H. Torney, as a model of its type. Exacting as were the responsibilities connected with this work, he found time to interest himself in the broader aspects of psychiatry, which were then demanding the best judgment and the most loyal devotion of the men who had visions of the future. The faulty laws, the lack of scientific interest, the apathy of the public and the fatalistic attitude of the medical profession as well as of the laity toward the treatment of mental disease were all matters which disturbed his sense of justice and which he forthwith set himself to rectify, so far as lay in his power. This public-spirited attitude led him early into a study of the question of "state care," which he strongly advocated. He made personal investigations of the condition of patients in almshouses, in which many of the insane were at that time housed without systematic effort toward their treatment, and had a large share in shaping

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public opinion and influencing legislation for the enactment of laws, which finally led to the exclusive adoption of "state care" for the insane. He was appointed by Governor Guild, in 1909, a member of a state commission to revise and codify the laws relative to the insane. As one of the results of this work, the mental condition of criminals came to be recognized as a fit and profitable study for the psychiatrist. A further measure to which he gave himself whole-heartedly was the possibility of developing a plan whereby certain of the harmless, chronic insane might be boarded out in families, thereby in some degree restoring them to normal life. It was very natural, therefore, when the movement of mental hygiene gained impetus that he should have been one of the first to see its importance and to help it toward gaining public recognition. He became a director of the executive committee of the Massachusetts society and was also identified with the national committee. In all of these movements for a better understanding of the problems of mental disorder and for a broader lay cooperation, he was a pioneer, untiring and courageous in the face of opposition and discouragement. Among his other activities he was for twenty years a trustee of the Taunton (Mass.) State Hospital, and one of the founders of the Monson State Hospital for Epileptics, which has become one of the recognized agencies for the study and treatment of this large group of dependents. It was a great source of satisfaction to him to see the accomplishment of the various reforms which he had sponsored and, in general, to have lived through the change of attitude toward the insane and their treatment which the last thirty years has developed.

Dr. Stedman was a forceful but not a voluminous writer; his papers were well considered and most carefully prepared, and concerned themselves largely with the measures to which he had devoted so large a share of his thought. Many of them dealt, as might be expected, with treatment. Articles on "after care," the "family system" and the "management of convalescence" are to be found among others dealing with various practical aspects of the general subject. A popular lecture on the "Mental Pitfalls of Adolescence," published by the Massachusetts Society for Mental Hygiene, was a noteworthy contribution to the society's propaganda.

He was a member of many medical societies, state and national. Among these were the American Psychiatric Association, the American Medical Association, the New York Psychiatric Society, the meetings of which he frequently attended, the Boston Society of Psychiatry and Neurology, and the American Neurological Association, of which he was president in 1906. He was a contributor to the programs of these societies from time to time, but a certain modesty and self-distrust prevented his participation on many occasions when it would have been welcomed by his fellow members.

In summarizing his life and accomplishment, one is impressed with the fact that he combined in unusual degree the capacity to divide his attention between his personal work and public affairs with no detriment to either. His almost over-conscientious devotion to his patients was the source of his professional success. Even after he had given up all personal responsibility, he continued to make regular visits to those who had been under his personal care, and the welfare of the hospital remained a matter of deep solicitude to the end of his life. Allusion has been made to his general activities, which will long stand as a monument to disinterested devotion to the public welfare. Appreciation of what he accomplished will not grow less with time. In his personal relations he was kindly, courteous, and a good friend to those in need of help. He took a very particular interest in young men starting in their professional life. His generosity was never paraded, and many acts of kindness, often of a material sort, were known only to him and to the recipients of his favors. He married Miss Mabel Weiss in 1879, who, until her death, in 1917, was an unfailing source of help and encouragement. A son, John W. Stedman of Morristown, N. J., and two daughters, Mrs. Albert Hale and Miss Anne Bradstreet Stedman, both of Brookline, survive him.

News and Comment

FIFTY-SECOND ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION

The next meeting of the American Neurological Association will be held in Atlantic City, June 1, 2 and 3, 1926, in the Hotel Ambassador.

THE NATIONAL ASSOCIATION FOR THE STUDY OF EPILEPSY

The next meeting of the National Association for the Study of Epilepsy will be held in New York in the Hotel Waldorf Astoria, June 7, 1926.

THE AMERICAN PSYCHIATRIC ASSOCIATION

The American Psychiatric Association will hold its next annual meeting in New York in the Hotel Waldorf Astoria, June 8 to 11, 1926, inclusive.

CORRECTION FOR ARCHIVES OF NEUROLOGY

An error occurred in the article by Dr. Hugo Mella, in the March issue. On page 330, third paragraph, the last word in line seven should read "highly," instead of "lightly."

Abstracts from Current Literature

HORIZONTAL PARALYSIS OF THE GAZE. THE OCULOMOTOR TRACTS. THE POSTERIOR LONGITUDINAL BUNDLE. A CLINICAL OBSERVATION FOLLOWED BY NECROPSY. ANDRÉ-THOMAS, Rev. d'oto-neuro-ocul. **2**:241 (April) 1924.

After reference to the importance of methodical study of these complex cases, both clinically and in microscopic sections after necropsy, the following case is reported: A woman, aged 62, had complained for a week of lack of power in the right limbs, difficulty of speech and swallowing, and visual disturbances. One year previously she had had severe epistaxis, hypertension and chronic interstitial nephritis. The illness began with headache, malaise and tingling in the right limbs, though power was not diminished; slight right-sided facial paralysis; abundant salivation, difficult swallowing; dysarthria. Anesthesia to pricking, heat and cold was present on the whole right side; the tactile sense was less altered; sense of position in the limbs was altered; the reflexes were exaggerated. The pupillary reflexes were normal; movements of the eyes upward and downward were intact, but no horizontal movement could be made; convergence was lost. The spinal fluid was xanthochromic and contained 5 cells per cubic millimeter. The blood Wassermann reaction was negative. After only slight improvement, coma and death occurred four weeks from the time of admission.

At the necropsy, a lesion of the pons was found, situated almost exclusively in the central part, more on the left side of the tegmentum, and extending from a plane passing above the origin of the sixth and seventh pairs of nerves down to a plane passing through the level of the commissure of Wernekinck. Serial sections were cut of the bulb, the pons, the peduncle and the corpora quadrigemina.

On the left side the superior peduncle had escaped, as had the lateral lemniscus and the external fifth of the median lemniscus; only a small part of the reticular substance was not destroyed. The structures involved were: posterior longitudinal bundle, the gray substance of the floor, the locus ceruleus, the small root of the fifth nerve, the circumflex nucleus of the reticular substance, the central fasciculus of the tegmentum, almost all the reticular substance and four-fifths of the median lemniscus. On the right, the lesion had destroyed the posterior longitudinal bundle, the locus ceruleus, the circumflex nucleus, the central superior nucleus and the adjacent reticular substance, and the internal part of the central fasciculus of the tegmentum; in the ventral part it was prolonged toward the internal part of the median lemniscus. In some sections it affected the trapezoid body and the internal edge of the superior olive on the left side, and sent a prolongation into the anterior layer at the level of the median raphe and of the most internal fasciculi of the left pyramidal tract.

We have then an extended lesion distributed in the territory of the median and lateral pontile arteries. It is perhaps primarily hemorrhagic, and the areas of softening are probably the consequence of anemia produced in the territory of the broken arteries. Hypertension has played an important part.

The roots of the fifth nerve showed partial secondary degeneration. Areas of secondary degeneration were found above the lesion in the posterior longitudinal bundle, median lemniscus and lateral lemniscus. The posterior longitudinal bundle on section has the shape of a comma with its round portion

placed medially, and between it and the median line are two or three narrow fasciculi called the "median fasciculi of the posterior longitudinal bundle." [NOTE: In this sentence author is describing normal anatomic configuration. T. L. D.] The tapering portion of the "comma" is placed laterally. The median fasciculi were not degenerated; the rounded part of the "comma" was markedly degenerated as well as the tapering part in its inner fifth, the outer part only partially.

The nucleus of the nervus patheticus is the first relay in which the fibers of the posterior longitudinal bundle terminate, and it is embedded in the bundle. At this level there is a close entanglement of the radicular fibers of the fourth and the terminal fibers of the posterior longitudinal bundle. The cells of origin of the nervus patheticus were embedded in a seed plot of black granules. The degenerated fibers of the posterior longitudinal bundle terminated also in the inner part of the nucleus of the third nerve.

The degenerated fibers of the median lemniscus could be followed in the median nucleus of Luys and beyond in the external nucleus of the thalamus. The degeneration in the lateral lemniscus could be followed on the left in the nucleus of the lateral lemniscus and up to the arm of the posterior corpora quadrigemina. On the right, the degenerated fibers stopped in the nucleus of the lateral lemniscus. The small root of the fifth nerve was not degenerated.

Below the lesion, the degenerations were localized principally in the posterior longitudinal bundle and the central bundle of the tegmentum, to a less degree in the pyramidal tract and in the median lemniscus. Some fibers were also found in the reticular substance, the trapezoid body and the descending roots of the trigeminus. Some fine degenerated fibers were disseminated in the nucleus of the sixth nerve on both sides, also in the triangular nucleus of the acoustic nerve and in the nucleus of the eminentia teres. No degenerated areas could be followed in the nuclei of the ninth and tenth pairs, and they were rare in the nucleus of the twelfth nerves.

The central bundle of the tegmentum was degenerated on both sides, totally on the right, extending to the olive. No degeneration appeared in the zone corresponding to the bundle of Helweg. Degenerated fibers were very rare in the inferior segment of the inferior central nucleus of the bulb, the superior segment being included in the lesion. On the right, degenerated fibers extended from the trapezoid body to the internal edge of the superior olive and another group occupied the reticular substance behind the superior olive. There was degeneration of a certain number of fibers which follow the fasciculi of the middle cerebellar peduncle, the white substance of the cerebellum, where they are again found in the external semicircular fibers.

Paralysis of the gaze in a single direction has been found many times in lesions of the pons, either pure or associated with paralysis of the sixth nerve, with or without permanent deviation to the side opposite the lesion. But bilateral paralysis is much rarer and requires a double lesion of the tegmentum, as in this case. Lateral paralyses, in either one or both directions, are characterized by the fact that the rectus internus, which does not contract in movements associated with the rectus externus of the opposite side, does contract in movements of convergence, consequently, in the movement associated with the contralateral rectus internus. Paralysis of convergence is encountered either alone or associated with a paralysis of gaze, especially a vertical paralysis. It is practically never associated with bilateral paralysis. In the case reported, convergence was nil and it seems better to speak of it as a horizontal paralysis, since all horizontal movement was abolished and only the vertical movements were conserved.

One finds in the literature some observations in which vertical and lateral paralyses are associated or combined, as, in some cases of vertical paralysis, lateral limitation of movement is mentioned. In analyzing the reports of lesions of the tegmentum in the neighborhood of the red nucleus and the nuclei of the third pair of nerves, one finds, with a paralysis of the third pair, a relative paralysis of lateral movements; but these are rather exceptional findings, and the lateral movements are only partially compromised.

Under the name of paralysis of the gaze a more complex syndrome is comprehended than appears at first sight. Recently these paralyses have been studied by analysis with regard to the physiologic conception, and different types have been distinguished leading to a better clinical, physiologic and anatomic conception. This is especially true of vertical paralysis, and it is remarkable that patients are seen who cannot raise or lower the eyes at the command "look up," "look down," but who can execute this movement when asked to fix the gaze on an object in the visual field or to follow a moving object; or in other words, when the patient reacts to an optic stimulus. There is occasion to study also what would be the reaction to the stimuli of acoustic, tactile, labyrinthine and neck reflexes. Investigation of caloric, galvanic and optic nystagmus, as well as movements of the eyes associated with closing of the lids, would be not less useful in these paralysis cases.

The lesion is differently situated, according as (*a*) there is loss of voluntary movement, (*b*) the reflex movements are partially or totally preserved, or (*c*) all movements, voluntary, reflex and associated, are abolished. In the first case the lesion does not affect the nuclei of the oculomotor nerves, but the absence of involuntary movements does not demonstrate a nuclear lesion. Preservation of involuntary movements points to a cortical lesion. Studies of lateral paralyses have been less exact.

In the case reported, the most important tracts involved in the lesion are the posterior longitudinal bundles and the reticular substance, because of the connections of the former with the nuclei of the third, fourth and sixth pairs of nerves. The posterior longitudinal bundle was not completely degenerated on either side above the lesion; the fasciculi between the median line and the compact part, and the inner side of the compact part itself, were not degenerated. The tapering part of the posterior longitudinal bundle was most degenerated in its inner side. Below the lesion, the degenerated fibers occupied the posterior longitudinal bundle and that part of the reticular substance between the median lemniscus and the compact fasciculus of the posterior longitudinal bundle. They are displaced as far as the level of the "collet" of the bulb where they abandon the posterior part of the posterior longitudinal bundle. At the level of the inferior extremity of the decussation of the pyramidal bundles, they occupied the anterior layer of the cord at a certain distance from the anterior horn.

In the case of Long and Roussy, the lesion was in the optic thalamus and the peduncular tegmentum, the latter being affected as far as the plane of the red nucleus and the decussation of the superior cerebellar peduncles. The descending degeneration is superimposed in part on that which existed in the case here reported, but is much less pronounced. If one compares the descending degeneration of the posterior longitudinal bundle in the case of Long and Roussy and the ascending degeneration in the case of the author, in the sections made at the level of the fourth pair of nerves, one will see that the degenerated fasciculi of the former are superimposed exactly on the nondegenerated fasciculi of the latter.

The posterior longitudinal bundle represents a system of ascending and descending association fibers of varying lengths which are not arranged by chance, but the fibers of a given origin occupy an invariable position. The descending fibers lose themselves in certain bulbopontile formations, such as the nuclei of the sixth pair of nerves, and the ascending fibers are largely distributed to the nuclei of the nervus patheticus.

The author has shown by degeneration experiments that a lesion of the pons in the region of the nuclei of Deiter and of the sixth pair of nerves causes ascending degenerations of the posterior longitudinal bundle which could be followed up to the nucleus of the third nerve of the opposite side, and a slighter area up to the third nerve nucleus of the same side. Below the lesion the homolateral fasciculus is more degenerated. He suggests that the fibers of the posterior longitudinal bundle, which take their origin in the neighborhood of the aqueduct of Sylvius and the nuclei of the third pair of nerves, establish relations between the corpora quadrigemina and the oculomotor nuclei, and that these two systems play an important rôle in certain ocular reflexes of vestibular or optic origin.

The case shows that there can exist bilateral or unilateral paralysis of the gaze without primary lesion of the nuclei of the sixth nerves. It is probable that, in the lateral gaze, the oculomotor nuclei are not directly subordinated to the others, but are synergically directed by one of several centers situated in the neighborhood of the nuclei of the sixth nerves, whose impulses travel through the posterior longitudinal bundle. An interruption of this bundle can explain isolated paralysis of the rectus internus in the lateral movement of gaze, while the same muscle functions normally in convergence.

One can admit the existence of fibers originating in centers in relation with the quadrigeminate bodies which go by a series of relays to the nuclei of the sixth nerves. In any event, it appears established that fibers originate in the region of the tegmentum, above the nuclei of the sixth and below the nuclei of the third which terminate in the nuclei of the sixth nerve. These nuclei receive also fibers from the vestibular nuclei, and perhaps other fibers whose origin is unknown.

If it is true that conjugate deviation of the eyes is clinically observed in hemispherical lesions, it is always only transient, whether the lesion is in the frontal lobe, the rolandic area, the angular gyrus or the occipital or temporal lobes. Lateral paralysis of the gaze is no longer mentioned in the syndrome of the thalamus or the central ganglia.

Vertical paralysis requires that the lesion approach the nuclei of the third nerves and be bilateral. For the production of lateral paralysis the lesion must descend still farther in the mesencephalon, in a vertical as well as in a horizontal sense. But it is an important fact that the integrity of the pathways whose interruption causes vertical paralysis is not indispensable to the synergy of lateral gaze. There exists a special descending tract for the lateral gaze, and it seems that conjugate movement of the eyes can be assured by either hemisphere.

The gaze can be commanded by an abstract representation: upward, downward, to the right, to the left, which action is different from the gaze that recognizes an object at the limit of the visual field, or follows a moving object; still more so, the gaze provoked by the sudden projection of an impression in the visual, the acoustic or the sensory fields; or the reflex associated movements produced by sudden brusk movements of the head and body (labyrinth and cervical reflexes).

If one remembers that the gaze is a movement, very often adapted to a sort of protection, one is less astonished to find in it an anatomophysiologic mechanism more complex and the pathways of realization less specialized. One could imagine that, in the lateral displacement of the ocular globes, the central excitation would follow either the pyramidal or the cerebellar tract (pyramidal tract, middle cerebellar peduncle, central grey nuclei, nuclei of vestibular nerve, nuclei of oculomotors). But in the patient here described this pathway was very little injured, and yet the bilateral paralysis was complete. Let it be remembered, though, that the tests were not repeated enough to demonstrate this point.

One will succeed more quickly in resolving the problem of paralysis of the gaze, if the function of gaze is thought of as depending on a physiologic mechanism, little specialized and localized, and if one considers also the considerable number of clinico-anatomic facts of a negative value; such as the cases in which the movements of gaze have been preserved either totally or in one or another direction, in spite of extended morbid lesions or their situation near the oculomotor nuclei.

This article contains so much of value in details of the microscopic examination and discussion of the pathologic physiology that no abstract can do it justice. It should be read in the original.

DENNIS, Colorado Springs, Colo.

THE PROBLEM OF LEFT-HANDEDNESS AND RIGHT-HANDEDNESS. W. KRAHMER and L. KORST, J. f. Psychol. u. Neurol. **31**:311 (June) 1925.

Wessely and his pupils (Köllner, Birnbacher and Dimmer) established the fact that the inner and outer halves of the retina of each eye are functionally heterovalent, i. e., that as far as the perception and localization of light is concerned, the inner retinal halves markedly exceed the outer. With this in mind, the present authors attempted to ascertain whether the functional heterovalence is limited only to heteronymous halves of the retina or whether it is also associated with homonymous halves (both nasal halves). To determine this, they conducted the following experiment, which is a modification of Wessely's original experiment in the sense that they could test both eyes at the same time. They constructed a special apparatus consisting of two metallic cylinders, each of which was completely closed, except for a 5 mm. wide aperture; the cylinders were freely movable over a bar. Inside of each cylinder, and on the same level with the aperture, a 5 candle-power electric lamp was so adjusted that both lamps could be lit or extinguished at the same time by means of a noiseless switch. The apparatus was set up in a dark room, 1 meter in front of the subject to be tested, who sat with his head immobile, and his ocular axes parallel to each other. Both cylinders were so arranged that the light from their enclosed electric lamps, emerging through the apertures in an opposite direction, at a distance of about 1 meter, would strike the subject's eyes at an angle of 60 degrees. The subject was instructed to describe, with every flash from the lamps, the light that he perceived simultaneously with both eyes, or separately with each eye.

Eighty-three persons between the ages of 22 and 40 were thus examined; they were all intelligent; sixty-six were Slavonians and seventeen Jewish. Among the latter there were seven men and ten women, whereas among the former the sexes were equally divided. Almost every subject was tested several times.

Of the thirty-three Aryan men, sixteen localized the light to the left, fourteen to the right and three once to the left and once to the right; of the thirty-three Aryan women, twenty-four localized it to the left, four to the right, four once to the left and once to the right and one ambidextrously, i. e., the subject saw both lights flare up with the same intensity, so that he could perceive no difference in intensity as between the right and left lamps; seven Jews localized it to the left, three to the right and two ambidextrously; six Jewesses localized it to the left, three to the right and one ambidextrously. According to these figures, the material could be divided into four groups: (1) one with a dominating functional light valence of the inner half of the retina of the left eye, a total of 57.8 per cent. of the entire number of persons tested; (2) one with a prevailing functional light valence of the inner half of the retina of the right eye, almost 29 per cent. of the entire material; (3) one group in which the persons were equivalent in the inner retinal halves of both eyes, 4.8 per cent.; (4) one group in which the subjects were either inexact in their answers, or could give no opinion as to what they saw, 8.4 per cent.

Disregarding the last two groups, it is obvious that the functional heterovalence of the inner retinal halves of both eyes must be regarded as a well established fact, in the sense that the functional prevalence of the inner retinal half of the left eye is at least twice as frequent as that of the right. This heterovalence, however, is essentially different from that established by Wessely, Köllner and others for the outer and inner halves of the retina. Wessely's experiments, dealing as they did with extremely old phylogenetic factors, showed that the heterovalence was stabile and did not change in any of the cases; the present experiments, however, in which new factors such as the constitution of the persons tested enter into the problem, show that the heterovalence is changeable, the functional prevalence being at one time in the left and at another time in the right half of the retina.

To approach the subject of heterovalence of the inner retinal halves on the basis of these new factors, the authors analyzed their clinical material from the following points of view: sex, national, individual, national-sex, national-individual and national-individual-sex characteristics with the right-handedness or left-handedness of the subjects tested as the basic principle of each group characteristic. From the figures obtained in this analysis the following conclusions are drawn:

1. Unilateral prevalence of light perception of the inner retinal halves does not depend as much on racial or sex characteristics as on the individual constitution. The figures show definitely that the prevalence of light perception of the inner retinal halves over the outer is to a greater extent, if not completely, dependent on right-handedness and left-handedness; it is characteristic for a right-handed person to localize light perception to the left, and for a left-handed person, to the right.

2. As far as racial characteristics are concerned, it may be said that Slavonians localize light perception to the left, whereas Jews localize it to the left as well as to the right. The ability of the latter to localize to the left is only slightly greater than their ability to localize to the right.

3. Slavonian and Semitic females equally localize light perception to the left. Left-sided light localization is characteristic for Slavonian males but almost unnatural for Semitic males, and conversely, right-sided localization is chiefly associated with Semitic males; it is strongly developed in Slavonian males and almost unnatural for Slavonian and Semitic females. In this respect, the females of both races resemble, as far as their individual characteristics

are concerned, persons with exquisite right-handedness; Semitic males resemble persons with exquisite left-handedness, and Slavonian males resemble the former as well as the latter.

4. Visual ambidexterity, i. e., the ability to perceive light equally with both inner halves of the retina, is an exquisite characteristic of the Semitic race. It is three times as frequent in Semitic males as in Semitic females, and it never occurs as frequently in Slavonians.

5. The relative number of left-handed persons is three times as great in the Semitic as in the Slavonian race, 75 per cent. of all left-handed persons among the Semites occurring in males.

The general impression obtained from these statistics is that there must exist some relationship between the direction of light localization and the individual characteristics of right-handedness and left-handedness on the one hand, and between the latter and the national sex characteristics on the other. This relationship is manifested by the fact that the direction of light localization and the characteristic of right-handedness and left-handedness is chiefly dependent on the individual, very strongly on the race, and possibly also on the sex characteristics. At the same time it must also be borne in mind that right-handedness and the ability to localize light to the left are characteristic for the females of both races, and to a certain extent for Slavonian males, whereas left-handedness and the ability to localize light to the right, as well as visual ambidexterity, are characteristic for the Semitic males. This being so, two questions present themselves for further discussion: 1. Does the ability to localize light depend on the characteristic of right-handedness and left-handedness, or is it a sequel to it? 2. How are the peculiarities as to light localization, dependent as they apparently are on race and sex characteristics, to be explained? In attempting to answer the first question, the authors divided all their right-handed and left-handed subjects who localized light exactly to the right and left into two groups, which they correlated, first, as regards right-handedness and left-handedness, and secondly, as regards their ability to react to light. The figures obtained showed that the difference between right-handed and left-handed persons as to light reaction was 64 per cent., whereas the difference between the light reaction in right-handed and left-handed persons was only 31.3 per cent. Consequently, it is not the light reaction, i. e., it is not the result of the impression of light on the retina, that determines right-handedness and left-handedness, but conversely, a person reacts to light perception in a certain direction only because he is either right-handed or left-handed.

In answer to the second question the authors point out that their statistics would seem to show that there may exist right-handed persons with left-sided light perception, as well as left-handed persons with right-sided light perception. This would justify the assumption that, in addition to the phylogenetic factors mentioned, there must exist in the problem of the direction of light perception some other factor which has in this respect impressed itself in some way on race and sex. Such a factor, they believe, might possibly be found in the handwriting of the various races. This assumption gains support in the fact that Semitic handwriting, undoubtedly invented by the males of that race, is constructed according to the characteristic of left-handedness, and is in full accord with the left-handed organization of the Semite. In the course of years, Semitic handwriting came to the Aryan races living near the Mediterranean Sea, especially to the Greeks, who, owing to their right-handedness, gradually remodeled the handwriting into a right-handed one. The Semites again living among the Aryans began to cultivate the hand-

writing of the latter, as a result of which they became in the course of evolution at first ambidextrous and later right-handed. Taking into consideration the origin and evolution of the handwriting of the Semitic and Aryan races as well as the fact that the females of both races are more right-handed than left-handed, one must attribute the fundamental cause of right-handedness and left-handedness to the cultural attainments of the various ancient races, which, although they depend to some extent on racial characteristics, are chiefly associated with the male element of the individual race. It was therefore the male who, thanks to his cultural achievement, also appropriated to himself the special constitution of right-handedness or left-handedness and disseminated it among his people.

It is generally assumed and rightly so, that a right-handed person differs anatomically from a left-handed one by the fact that in the former the left cerebral hemisphere predominates over the right, and in the latter, the right over the left hemisphere. Clinically, this fact is amply substantiated by the manifestations of motor aphasia. The present investigation, however, would seem to show that primitive light perception as well as light localization in right-handed persons is associated with the right hemisphere and in left-handed persons with the left hemisphere. If this be so, the anatomic organization of right-handed as well as of left-handed persons must be much more complicated than has hitherto been believed. It is likely that both hemispheres participate functionally in right-handed as well as in left-handed persons. In the right-handed, as far as motility is concerned, the left hemisphere, and as far as primitive sensory centers are concerned, the right hemisphere predominates, whereas in the left-handed the opposite of this occurs.

In closing, the authors state that owing to the paucity of their material and to the possibility of error in their experiments, their conclusions on this problem need not be considered as the last word on the subject. They are publishing their work for the purpose of stimulating further research in this direction, and they also believe that eventually cyto-architectonic studies will either completely confirm or totally deny the truth of their hypothesis.

KESCHNER, New York.

DIE REVISION DER NEUROSENFRAGE. OSWALD BUMKE, München. med. Wchnschr. **72**:1815 (Oct. 23) 1925.

Bumke in his usual classic manner discusses first the historical aspect of the neuroses. He states that in the latter part of the eighteenth century, Cullen, a Scotchman, first used the term neurosis in a very loose way to designate innumerable affections. M. Romberg, in 1840, included under this heading all peripheral, spinal and cerebral palsies, also tabes and general paralysis. It was during this time that the term reflex neurosis was developed, which, as so many other terms, gradually vanished although even today it is not unusual to see a woman who has had her ovaries removed as a result of so-called hippocratic reflex hysteria.

With the impetus of Virchow's work an entire revision of nervous affections took place. About 1870, the neuroses constituted nervous disorders for which no pathologic anatomic cause was known. Erb, in his classic book published in 1874, still included under the caption of the neuroses numerous peripheral nerve affections, tetanus, paralysis agitans, etc. He appreciated, however, that organic and functional conditions merged into one another and that in due course of time their relationship would be better established.

Following the work of Erb and others the psychologic foundation for functional diseases gradually developed. The chief advocates at this time were Charcot and then Janet. Such a profound student as Oppenheim, who was reared on the pathologic cause for disease, continued at all times to assume an organic basis for functional diseases. In the war neuroses, even though the microscope failed to show any pathologic changes, he clung to such explanations as concussion, diaschisis, etc., as the organic basis of the ailment. It is Bumke's conception that Oppenheim as a result of his training, conceived the brain when one used the term soul or mind of the neurotic and that he could not entirely leave his organic background for a psychologic conception.

In 1880 Beard presented to the profession the word neurasthenia. Probably no word in the history of medicine produced such a turmoil. It was not long before most functional conditions and even the mild psychoses were classed under this heading. Under the guidance of Forel, Bleuler and Dubois, the pendulum swung back so that in 1894 Charcot was able to write as a foreword in Janet's book that hysteria was a distinct psychic disturbance. Janet developed his psychasthenia, elaborated on hysteria and separated hypochondria and neurasthenia; then came Freud's numerous contributions.

During this entire evolution there remained an undercurrent of organic, somatic, humoral and reflex theories. Nissl, in 1902, took the stand that hysteria must have an organic basis just as has general paralysis. This, however, he could not demonstrate. Hoche about this time, in discussing the differential diagnosis between epilepsy and hysteria, stated that hysteria as yet possessed no pathologic anatomy in the sense of general paralysis and that it could not and never would.

Following Bumke's reasoning it should be clear that all organic conditions are prone to functional factors. When the spirochetes wander to the brain, when a tumor grows, when a hemorrhage occurs or a severe injury is suffered, there must also occur a psychologic factor; for in all these conditions the natural psychic qualities are changed. To quote the author still further, it is as if a child grasped with outstretched hands into the mechanism of a clock.

It may, indeed, be correct that functional conditions have as a basis some bodily change, whether dynamic or chemical must be left an open question. Bumke is emphatic in his criticism of the present day tendency to utilize new words for the expression of new ideas concerning which so little is known. Much is written about the sympathetic, vagotonic and endocrine anomalies about which we all know so little; and would it not be better to have patience and wait for help from the physiologic chemist?

As individuals vary, so do functional nervous disorders vary. There is no well defined pattern to which they always conform. In the organic nervous disorders it is quite different, and one may formulate very exact classifications. At one time there was a disease hysteria, hypochondria, etc. As isolated diseases they have disappeared, and we now recognize them only as certain types of reaction, constitutional, developmental or even resulting from some hereditary influence.

It must be clear that in every neurosis the factors of constitutional make-up, as also acquired causes, must be investigated. In other words, symptoms may be based on bodily changes as well as on psychic changes. As stated previously, psychogenic symptoms may be observed in alcoholism, general paralysis, brain tumors, senile dementia, etc. During the war it was observed that month by month greater numbers of functional cases developed as a result of under-

nourishment, loss of sleep and increasing infections, showing that bodily changes must predispose to functional conditions.

Not infrequently the patient's attention is called to a certain organ as a result of bodily difficulties, and a psychogenic halo may develop about the organ. Later there develops a psychogenic disturbance which, however, had its inception in a true somatic ailment. The most simple voluntary movements are as yet not entirely explainable. We know that when we wish to raise an arm and the arm really goes up that something has occurred in the brain to produce this action. This we all call physiologic, but, after all, this does not entirely explain the situation. It is this same fundamental problem that we have to deal with in functional conditions such as in hysterical motor phenomena.

Just why certain individuals adopt certain reaction types is not clear. No explanation seems to serve in all cases for a clear understanding of the problem. Bumke again emphasizes that his chief opposition to psychanalysis is not the sexual motive but the naive method it adopts in explaining everything and in always being correct. This tendency to confusion in psychanalysis is most deplorable, and the author cannot see that we are getting anywhere in our present method of dealing with the subconscious.

Bumke emphasizes that he fully realizes the importance of our subconsciousness. Every psychic process originates there and leaves its imprint there. He does not believe, however, that our subconscious dominates all our conscious acts in their entirety, as one might be led to believe by the psychanalyst. According to Bumke, the psychoneurotic group is being replaced by reaction types such as nervous reactions, nervous constitutions, the psychopathies and the functional psychoses. The term hysteria, however, will live on for a long time. The neuroses with their mixture of physical and psychic qualities, as is also the case in the manic-depressive psychoses, will naturally continue to exist. Certainly it is true that with the advance in neurology and psychiatry, the neuroses are gradually being confined within narrower and narrower limits.

MOERSCH, Rochester, Minn.

A CASE OF ANGIOMA ARTERIALE RACEMOSUM IN THE REGION OF THE RIGHT MIDDLE CEREBRAL ARTERY. WOLFGANG LAVES, Jahrb. f. Psychiat. u. Neurol. **44**:55, 1925.

Laves could find only fourteen cases of this condition reported in the literature. His case occurred in a woman, aged 51, who had been suffering from gonorrheal pyosalpingitis since 1907, for which she was finally operated on in 1921. She had given birth to two children. There were no miscarriages and no history of syphilis and the blood Wassermann test was negative.

In 1912 the patient began to have attacks of twitching in the muscles of the left hand; these attacks were not associated with disturbances of consciousness but were accompanied by a loss of sensation in the entire left half of the body. At the onset the attacks would appear several times a day. During 1916 and 1917 they were associated with nausea, vomiting, fever, diarrhea, pain in the right side of the body and loss of consciousness. Simultaneously with these attacks she began to have twitchings in the left leg and arm. She had six such attacks up to the fall of 1922. In November of the same year, after drinking two glasses of wine, she suddenly became disoriented and nauseated, and complained of severe occipital headache. Soon thereafter she collapsed and became unconscious. On admission to the hospital

she slowly regained consciousness and began to complain bitterly of pain in the back of the neck; she vomited repeatedly, and stated that she felt as if her left arm were "dead." Examination revealed tenderness and rigidity of the back of the neck; the right eyeball was turned to the right; she appeared somewhat emaciated; percussion of the right side of the skull gave a tympanitic note; there was marked pressure tenderness over the first and second branches of the right trigeminus and over the right occipital nerve; the left upper extremity showed a definite paresis with astereognosis and ataxia; the abdominal reflexes were absent, the patellar reflexes were diminished on both sides; there was a bilateral Babinski sign, and while being tested for a Romberg sign she swayed backward. Her psyche was intact. She complained continually of headache and was constantly yawning; there was also a slight rise in temperature. All these manifestations disappeared within the next few days. Operative intervention was advised but refused. She remained well for eight days, after which she suddenly developed severe pains in the head, the small of the back and the back of the neck, with continuous convulsive twitchings of the left side of the face and of the left upper extremity, without loss of consciousness. The next day she showed marked prostration, a left hemiparesis, rigidity of the neck and some tenderness on pressure over the entire back. She presented a clinical picture of meningitis. Soon there were also papilledema, retention of urine, difficult deglutition and tenderness of the skull on percussion. Her pulse was very rapid. Gradually these symptoms also began to abate in severity; her pains diminished, her sensorium became clear, and she began to recover the use of the left arm. On Dec. 25, 1922, Professor Hertle performed a craniotomy, exposing the right central convolution and the adjoining parts of the temporal lobe. On opening the dura, the brain was found to be nonpulsating and covered with a network of dilated vessels, apparently veins; arterial pulsation could not be observed in these vessels. On introducing a needle 2 cm. below the cortex, the syringe immediately became filled with blood; after withdrawing the needle, a stream of blood came gushing out through the puncture hole. Further surgical intervention seemed inadvisable; the patient was in poor condition, so that only a few vessels could be ligated. She died two days after operation.

Necropsy showed in the posterior central convolution and in the region adjoining the inferior parietal lobe a sharply outlined, spongy, vascular coil the size of a plum; this tumor consisted of numerous vessels, most of which were arteries of varying size. A small artery was seen emerging from the fissure of Rolando, probably a large branch of the middle cerebral artery, which could be traced into the tumor mass; from the middle and posterior portions of the tumor two veins were seen emerging and emptying into the middle cerebral vein. The adjoining cerebral veins were markedly dilated; the pia was congested. There was also a perforation into the right lateral ventricle. Microscopic examination revealed dilated arteries, veins and capillaries. The principal changes in the intima of the arteries were in the form of spigot-like and flattened thickenings with a marked increase in the elastic elements; the adventitia and media showed considerable variations in thickness. The veins were also markedly dilated and showed evidences of retrogressive changes. These findings established the diagnosis of angioma arteriale racemosum in the region of the right middle cerebral artery with a perforation into the right lateral ventricle.

There is no unanimity of opinion as to the genesis of these angiomas. Most of the cases, according to Heine and Schück's statistics, seemed to have

originated from congenital telangiactases. Borst believes that the difficulty was due to an overdevelopment of the fetal anlage of these vessels. Virchow speaks of the relationship of these vascular coils to the branchial clefts (angioma fissurale); von Rindfleisch attaches great significance to partial disturbances in the vasomotor nervous system, and some authors attribute a causal relationship between trauma and tumor formation.

The literature permits the three following classifications of angiomas: (1) cases without characteristic changes in the vessel walls, (2) cases in which hypertrophic processes in the vessel walls predominate over degenerative processes, (3) (one case) cases in which the process consists solely of a degeneration of the vessel walls. The case reported in this contribution is included by the author in the second group. A review of the cases reported in the literature appears to indicate that the clinical picture of the condition depends, to some degree, on factors which tend to hasten the development of these angiomatous growths by virtue of a transitory or continuous increase of blood pressure. Some of these factors are cardiac hypertrophy with palpitation, exophthalmic goiter and hormonal influences (Virchow, Heine and others), such as the onset of puberty, menstruation and pregnancy, with the unusual demands that these conditions make on the cardiovascular system. Other factors are increased blood pressure due to physical overexertion and alcoholic and sexual excesses.

The paper concludes with a discussion of the relationship between trauma and the formation of angiomas. The attempt to harmonize the pathologic findings as to the site of the lesions with the anamnestically obtained data as to the area involved in the trauma does not justify the conclusion that trauma can be regarded as a direct etiologic factor.

KESCHNER, New York.

BILATERAL AND COMPLEX BULBAR SYNDROME IN SYRINGOBULBIA. REMARKS ON DISTURBANCES OF THE VESTIBULAR APPARATUS AND ON THE OCULOCARDIAC REFLEX. J. A. BARRÉ and R. CAHN, Rev. d'oto-neuro-ocul. **2**:271 (April) 1924.

This study is of a man, aged 21, who was healthy up to April, 1922. Without apparent cause he began to have sensory disturbances (thermanesthesia and analgesia) in the right hand and arm. Following these symptoms, which pointed to the cervical region of the cord, there arose a series of bulbar troubles, which suggested an extension upward of the process.

On examination there were found: atrophic paralysis of both vocal cords; paresis and fibrillation of the left half of the tongue; paralysis of the soft palate; paralysis of the right constrictors of the pharynx with abolition of the gagging reflex; left facial paresis with spasm of the orbicularis, accentuated by lateral and upward movement of the eyes; conservation of the oculocardiac reflex on both sides; vestibulo-ocular disturbances; total absence of signs of cerebellar disturbances; irritation and deficiency disturbances of the pyramid on the right; hypothermy of the left face. The spinal fluid was normal except for a slight lymphocytosis (6 cells). The Bordet-Wassermann reaction was negative in the blood and spinal fluid.

The authors make special mention of the vestibular disturbances, which came on at a definite period; the initial manifestation was frank lateropulsion to the right. The findings were: spontaneous nystagmus to the left; deviation of the arms to the right; inclination of the body to the right and falling to the right. In the caloric test: when the right ear was irrigated with 40 cc. of water at

27 C. (position of head not stated), horizontal nystagmus to the left and deviation of the body and arms occurred to the right; when the left ear was irrigated with 50 cc. of water at 27 C., there was horizontal nystagmus to the right. With 200 cc. of water, there was violent nystagmus and slight deviation of the arms to the left, with falling to the right.

Turning Test.—With the patient sitting, after ten turns to the right, a few twitches of eyes with divergence occurred, and the patient felt discomfort. After ten turns to the left, violent nystagmus (kind not stated) to the right occurred and lasted ten or twelve seconds; there was no discomfort, divergence of eyes or inclination of the head. On the turn table, with dorsal decubitus, head straight, and turn to the left, curving of the upper half of the body and rotation to the left occurred during the turning; when the table stopped after ten turns, horizontal nystagmus to the left occurred. After ten turns to the right, there was no nystagmus or head movement. With dorsal decubitus, head turned to the right, ten turns to the left, there was horizontal nystagmus to the right for five seconds; after rotation ten times to the right, with the head turned to the left, there was no nystagmus. (The authors claim that in the first of these two latter positions the left labyrinth is excited, and in the second the right is stimulated. The abstracter cannot comprehend this statement.)

The galvanic test revealed inclination of the head to the right (positive pole on left ear) even with 15 milliamperes of current. The negative pole on the left ear accentuated the spontaneous falling to the right with 8 milliamperes of current. When the negative pole was placed on the right ear, the head began to turn to the left with 3 milliamperes, reached the midline with 10 milliamperes and passed to the left with 15 milliamperes. Nystagmus occurred with 2 milliamperes of current in both directions. No report of an examination of the hearing is given.

Although there was no cerebellar disturbance, the patient walked with spread legs; the authors state that they have seen this at times in pure vestibular affections. The patient had never had the least vertigo, although he had had numerous subjective and objective signs of lesion of the vestibular tracts. The authors ask if a relation does not exist between the absence of vertigo on the one hand and the central seat of lesions and their evolution, probably slow, on the other, which could be later used in the topographic diagnosis of lesions of the vestibular apparatus. The nystagmus to the left, deviation of arms and body to the right and falling to the right point to a lesion of the right vestibular apparatus. To these signs may be added the unilateral voltaic deviation to the right and the falling to the right with the caloric test. (It should be noted that the falling is to the right even when the left ear is being douched with cold water: also, that the nystagmus from stimulation of the vertical canals of both sides is perverted, although the position of the head is not stated.—*Abstracter.*) The authors say, "Perhaps one can consider all these phenomena as allied to a bulbar disturbance of the vestibular nerve or an alteration affecting particularly the system of fibers coming from the horizontal canal." Another fact that indicates a lesion of the vertical canal fibers is that the horizontal nystagmus produced by the caloric test could not be made rotatory by a bending of the head to the shoulder opposite the ear irrigated. They say, if we accept the significance of this fact, that the vertical canals are inexcitable, we must admit either that the lesion is bilateral, even though we must believe it unilateral up to now, or that a unilateral lesion of vertical canals can cause an absence of excitability of the other side, as Bárány suggests. They draw a comparison

between the phenomena of intrabulbar lesions and those which are considered by American authors as peculiar to solitary lesions of the acoustic nerve, and ask if we are justified in regarding the symptom-complex of inexcitability of both horizontal and vertical canals of one side, together with loss of reaction of the vertical canals of the other, as indicative of pressure on the vestibular nerve by acoustic tumor on the side with total inexcitability. (The angle lesion symptom-complex of Jones and Fisher is loss of hearing and reactions from all canals of one ear; loss of reaction of vertical canals, but a good reaction from the horizontal canal, plus no disturbance of the cochlear branch, of the opposite ear.—*Abstracter.*) Granting that unilateral lesions of the horizontal canals or their nerve fibers ordinarily give rise to unilateral disturbances of reaction to stimuli, they ask if there may not be much greater differences between the fibers of the two groups than have been believed. And they suggest that these differences can concern either the pathways in the brain stem or perhaps, especially, the reciprocal connections of the two systems of fibers; those of the horizontal canals remaining independent and those of the vertical canals (of the two sides?) forming a sort of functional and anatomic unity by reciprocal connections.

DENNIS, Colorado Springs, Colo.

THE AMYOSTATIC SYNDROME. OTTO MARBURG, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **27**:47 (May) 1925.

The regulation of movement depends on two mechanisms: one, proprioceptive and labyrinthine from the periphery, and another which is associative from the cerebral cortex. Between these two are inserted the cerebellum and basal ganglia, both of which receive impulses from the cortex and from the periphery, and after rearranging them, transmit them to the muscles. From this point of view the structure of both of these organs may be said to be identical. They consist of a number of smaller cells, designated as nuclei in the cerebellum, which receive the proprioceptive, labyrinthine and cortical impulses, transmitting them to the large cells, which may be designated as "collectors," because they collect the various impulses as they reach them. After being thus "collected" the impulses undergo further condensation by nuclei which may be regarded as outposts of the receptive centers, i. e., the dentate nucleus in the cerebellum and the globus pallidus in the striatum. In this connection it must also be emphasized that the striatum receives the impulses by way of the thalamus. As movements in general become more numerous, more complicated and more delicate, these two portions of the brain begin to grow—the neocerebrum being added to the so-called pallium. The actual motive for this arrangement is the fact that in the higher vertebrates all movements are associated with the cerebral cortex, whereas automatic movements regress completely. This would seem to indicate that the higher we ascend in the animal scale, the greater is the transformation of the subcortical reflex centers for automatic movements. The large motor cells diminish in number and are replaced by an increased number of smaller cells, which eventually receive the impulses to transmit them further, without much change. This is best illustrated by the nucleus ruber and the nucleus interstitialis.

In this manner the impulses reach the cerebral cortex which is of such predominating importance in the adult that the subcortical centers, of greatest significance in childhood, as far as movement is concerned, entirely recede. The cerebellum has a centripetal connection with the cerebrum by way of the brachium conjunctivae, but the striatum and pallidum have no such connection;

one may therefore assume that pallidal impulses also reach the cerebrum by way of the red nucleus and the brachium conjunctivae. It is difficult to evaluate the significance of these two centers; they cannot be regarded as being directly opposed to each other. Thus we find hyperkinesias, although rarely in lesions of the cerebellum and asthenia as well as disturbances in tonus, in lesions of the latter, as well as in those of the striatum. Therefore, all that one can say is that we are dealing with an apparatus for coordination which phylogenetically consisted originally of a few extensive ganglionic "anlagen." Originally the cerebellum regulated only muscles subserving locomotion by determining the size of the movement, the degree of tonus and the amount of power of the muscles engaged in movement. The function of the pallidum was to "round" out the movement and to assure its maintenance, i. e., to influence posture. That tonus components play such a predominant rôle in this mechanism is due to the fact that tonus has by far greater significance for the maintenance of posture than any other element related to it. In the course of growth, as the mechanisms for movement became more extensive, especially as they became connected with the cerebral cortex, the regulating mechanisms also became connected with it, so that both of these mechanisms could exert their influence over the entire musculature of the body, from one central source. Marburg, therefore, looks on these so-called extrapyramidal motor disorders as disturbances in the regulating mechanisms — centripetal and not centrifugal disturbances of innervation.

The complicated part in the mechanism of movement is not the transmission of impulses from the center to the periphery. This is unusually simple; the impulses are transmitted from the cortex by the pyramidal tracts, from the mesencephalic centers by the rubrospinal system and the other descending fibers in this region. More complicated, however, is the mechanism regulating movement; the slightest disturbance in this mechanism has some effect on movement, the cause for which must be sought in a lesion involving centrifugal motor pathways, pyramidal, as well as extrapyramidal. Clinical and experimental, as well as anatomic, evidence, however, does not bear out completely the truth of such a conception. The designation, therefore, of extrapyramidal movements as such, seems to be less justified than their designation as "amyostatic syndrome" — a designation which does not necessarily limit disordered movement to the extrapyramidal system, as the former designation would seem to imply. The clinician can obviate this difficulty by designating each syndrome by a term that would express the nature of the most significant feature which the case in question presents clinically.

KESCHNER, New York.

A STUDY OF FOUR HUNDRED AND FIFTY CASES OF EPIDEMIC ENCEPHALITIS. JOSEPHINE B. NEAL, HENRY W. JACKSON and EMANUEL APPELBAUM, Am. J. M. Sc. **170**:708 (Nov.) 1925.

The cases were studied during the period between the fall of 1918 and the beginning of 1924. The authors had been studying poliomyelitis, meningism, meningitis and other acute infectious diseases involving the central nervous system previous to this time. Their results in encephalitis are presented in five tables, the first showing a greater number of cases during the first three months of the year and the smallest number during the summer. Fifty-nine per cent of the patients were males, and 41 per cent females. In table 3, a study of age distribution shows 217 patients were less than 15 years of age. Though they saw many children, they believe the diagnosis is often missed in this class of cases.

The symptomatology was varied. The virus may involve any part of the central nervous system and may progress from one part to another during the course of the disease. The onset was usually gradual, extreme lethargy and somnolence being quite characteristic when present, but restlessness and insomnia were also common; physicians who look for lethargy, asthenia and cranial nerve palsies will err in many diagnoses. Ocular disturbances were frequent, and include diplopia, blurring, ptosis and occasionally temporary blindness. Twitching, torsions and deviation of the head and eyes were sometimes present. A normal fundus was the rule, but hazing and even choking of the disks were observed. Headache was almost constant. Lancinating pains in the extremities were frequent, pains in the thorax less common. Trophic disturbances, especially ulcers of the cornea were noted rarely. Hypertonicity of the muscles of the face, neck and trunk was frequent. Weakness of the muscles of deglutition was seen. The reflexes were diminished, normal or exaggerated; Babinski, Brudzinski and Kernig signs were often noted. Vomiting was frequent, while Cheyne-Stokes breathing was rare except in the terminal stages. In children the disease was milder and more atypical, and more closely resembled tuberculous meningitis.

The differential diagnosis from tuberculous meningitis may be made with certainty from the spinal fluid, but clinically is very difficult. The encephalitic form of poliomyelitis is probably epidemic encephalitis and the classification will have to be revised. The differentiation from tumor, cerebral hemorrhage and thrombosis, uremia and certain forms of mental disease is also difficult.

The results of blood examination are not characteristic; usually there is a low leukocytosis with from 10,000 to 15,000 cells. The cells in the spinal fluid are ordinarily less than 100 with a preponderance of the mononuclear type. Rarely, an increase of cells up to 1,000 may be seen. There is an increase in globulin and albumin. The spinal fluid sugar varies from 50 to 100 mg. per hundred cubic centimeters. The chief value of the sugar study is to differentiate the condition from tuberculous meningitis in which the sugar is consistently low. In a few instances the fluid is normal, but these observers believe such a report usually signifies an incomplete examination.

The pathology is that of an inflammatory disease and may be found throughout the nervous system. There are four kinds of lesions: infiltration of the walls of the small vessels with lymphocytes and plasma cells; foci of interstitial and parenchymatous infiltration with round cells; lesions of the nerve cells, usually in an inflamed area but occasionally elsewhere; foci of perivascular hemorrhage. No treatment is of special importance. Repeated lumbar puncture seems to be most beneficial.

Table 4 shows a mortality rate of 28.2 per cent, 68 per cent for the fifth decade. The disease is now considered to be chronic with frequent remissions and exacerbations. Conditions formerly considered sequelae are now thought to be secondary attacks or relapses. These may follow the acute stage in a definite progression.

Of the 450 patients, 127 died. Of the remainder, 288 have been investigated, and seventy eight or 27 per cent show sequelae while the others are apparently well. The greater number of those showing sequelae were over 15 years of age. The greatest number of sequelae are mental and the second greatest motor. Only eleven cases showed a parkinsonian syndrome. Paralysis or weakness of some muscles of the extremities is the most common motor finding. The sequelae, therefore, are varied, and differ from those of poliomyelitis in which flaccid paralysis is found and from meningitis which has comparatively few.

WILSON, Clifton Springs, N. Y.

THE THEORETICAL SIGNIFICANCE OF SUBOCCIPITAL PUNCTURE. ADOLF HARTWICH, Deutsche Ztschr. f. Nervenh. **85**:21 (March) 1925.

According to Sicard and Nonne, the composition of the spinal fluid is the same throughout the cerebrospinal system. Fisher, in a thorough histologic study of twenty general paralytic patients, has found a definite relationship between the cellular elements of the spinal fluid and the degree of infiltration of the meninges in the lumbar region. This relationship does not exist in the cervical region or in the meninges of the brain. This is not in accord with the observations of Alzheimer, Plaut, Meyer and others, who believe that the meningeal infiltration does not influence either the number or type of cells in the spinal fluid.

In a study of twenty-four spinal fluids (twenty general paralysis and taboparesis) Walter noted that in fractional examination of the fluid from the lumbar region the first portions gave a higher cell count. This has been confirmed by many workers. Weigeldt and others have found a constant variation in the cell count of normal spinal fluid at various levels. The fluid from the ventricles was free from cells while that from the lumbar region contained the greatest number. In pathologic fluids the cellular increase depended entirely on the character and location of the pathologic process and on the sedimentation of the cells. In a study of fifteen normal spinal fluids the author found in 75 per cent of the cases a somewhat higher cell count from the lumbar than from the suboccipital puncture. In only three of nineteen pathologic spinal fluids was there a slight variation, while in the remaining sixteen a marked difference was noted at the two levels. In most cases the greatest number of cells was found in the fluid from lumbar punctures. However, in four cases of epidemic and tuberculous meningitis, the cisternal fluid gave much greater increase. Fractional examination of the spinal fluid from the lumbar puncture in these cases showed that there was a gradual increase in the number of cells; the first portion gave 129 cells, the last, 392 cells. Fractional examination of the cisternal fluid gave a higher cell count in the first fluid drawn. This is probably due to the fact that after a certain amount of cisternal fluid is withdrawn ventricular fluid is obtained. In several cases of tuberculous meningitis the last portion of the cisternal fluid gave a greater cell count. This the author believes to be due to a marked inflammatory reaction of the choroid plexus.

The author fully discusses the cerebrospinal fluid circulation. He points out that the fluid is secreted by the choroid plexus, flows through the foramina of Magendie and Luschka into the spinal subarachnoid space and is there absorbed. The increased intracranial pressure and the gradual absorption of the fluid in the lower spinal sac produces a slow, constant downward flow of the fluid with a tendency for cell sedimentation in the lumbar portion. This flow is accellerated by the circulatory and respiratory waves, by increase of intracranial pressure caused by coughing, and by motor activity. A case of dementia praecox is reported in which during the catatonic stupor the cell count in the lumbar fluid was much greater than in the cisternal fluid, while during a maniacal phase of three weeks' duration the cell count at the two levels was practically the same.

According to the author's observation, the circulatory and respiratory waves and muscular activity play a greater rôle in the spinal fluid circulation than the intracranial pressure. Dyes diffuse as rapidly through the spinal fluid whether the injection is made through the suboccipital or lumbar region, and whether the patient is in the erect or recumbent posture. Coughing and

muscular activity hasten this process while suppression of circulation and respiration and complete relaxation, such as is produced by a large dose of morphin, delay the flow of spinal fluid. In a patient almost pulseless and with very shallow respiration, there was practically no admixture of spinal fluid with the dye solution.

HAMMES, St. Paul.

LA DÉGÉNÉRESCENCE HEPATO-LENTICULAIRE. ETUDE CLINIQUE, ANATOMIQUE ET EXPÉRIMENTALE. IVAN MAHAIM, Schweiz. Arch. f. Neurol. u. Psychiat. **16**:252, 1925.

About the time Wilson established the morbid entity of progressive lenticular degeneration (1912), another affection, similar in nature, became an entity through the anatomic-pathologic contribution of Alzheimer. This disease, known as pseudosclerosis, was recognized clinically since its description by Westphal (1883), Francotte (1887) and Strümpell (1898). At that time it was stated that these cases suggested peculiar types of multiple sclerosis. Hall, in 1921, published a complete review of contributions dealing with Wilson's disease and with pseudosclerosis. He believed, further, that these cases were related to torsion spasm, and included all of them under the caption hepatolenticular degeneration. Clinically and anatomically they have much in common. The symptoms of Wilson's disease and pseudosclerosis are much alike. Both exhibit extrapyramidal motor disturbances, tremor and hypertonia. In pseudosclerosis the tremor is more prominent, and in Wilson's disease the hypertonia is more manifest. In both affections dysarthria, dysphagia and disturbance of gait are the result of hypertonia. The pyramidal tracts and sensation are not involved. Mental disturbances are present, particularly in pseudosclerosis. Pigmentation of the cornea, when it exists, is pathognomonic of these two types, but is more frequently observed in pseudosclerosis. A familial appearance is noted in 50 per cent of cases. In spite of the advanced cirrhosis of the liver, there are few symptoms from it. Alimentary levulosuria appears to be rather constant. In pseudosclerosis phosphaturia is often marked. The spleen is often hypertrophied.

Nor are the pathologic findings in the brain distinctive. Alzheimer found no macroscopic lesions in the brain in pseudosclerosis. He described the degeneration of ganglion cells and changes in the neuroglia, particularly giant cell formation in the gray matter of the basal ganglia. In Wilson's disease, on the other hand, the changes are often macroscopic, and cavitation may take place. The putamen of the lenticular nucleus is usually involved. A proliferation of the neuroglia occurs. These changes, however, represent extremes in both directions and many cases occupy an intermediate ground. Changes in the liver are the same in the two conditions.

A third disorder, torsion spasm, appears to be a member of the same group. It was at first classified among the neuroses. Oppenheim believed that an organic change must underlie this disorder, in spite of the negative results of one necropsy conducted at that time. The disease begins between the ages of 10 and 20 years. The onset is insidious; there are cramps, clonic spasms and bizarre involuntary movements of torsion, which involve the limbs and trunk but do not affect the face. It was the alternation of hypertonia and hypotonia that led Oppenheim to designate this disturbance as dystonia musculorum deformans. There are no changes in the psychic sphere. It was chiefly through the pathologic findings that the relationship of this disturbance and the two other diseases discussed above became apparent. Here too, focal

lesions in the lenticular nuclei and cirrhosis of the liver are found. The fact that Thomalla's case was not considered altogether typical of torsion spasm, since it also had some of the features of Wilson's disease, simply serves to emphasize the close relationship that exists among these diseases. Thomalla's case showed the dysarthria and dysphagia of Wilson's disease, as well as torsion spasm. No tremor was present. The necropsy disclosed cirrhosis of the liver, large degenerative lesions of the lenticular nucleus, principally of the putamen, and some changes in the body of Luys. Wimmer's case likewise presented difficulty of speech and deglutition and torsion spasm, but no tremor and no corneal pigmentation. (*To be continued.*)

WOLTMAN, Rochester, Minn.

FURTHER INVESTIGATIONS ON THE INFLUENCE OF THE MALARIAL TREATMENT OF GENERAL PARALYSIS ON THE HISTOPATHOLOGIC PROCESS. E. STRÄUSSLER and G. KOSKINAS, Ztschr. f. d. ges. Neurol. u. Psychiat. **97**:176 (June) 1925.

This article from the clinic of Wagner-Jauregg is based on a study of thirty-eight cases with death during the course of malarial treatment. Of these patients, four had a rise in temperature but no malaria, three had less than five malarial attacks and thirty-one had more than five malarial attacks. The important question was whether there existed a parallelism between the remissions produced by malaria and the pathologic picture, having in mind the opinion of Alzheimer that such a parallelism does not exist. On the basis of three cases previously studied and reported, the authors state definitely that "with remissions in the clinical picture there exists a regression and a stationary condition of the pathologic process." This view is fully substantiated by the pathologic studies of other cases. The authors describe in detail a case of a patient with general paralytic dementia, first treated with malaria in 1921, who developed a good, but not complete remission. Three years later this man died, following an operation for adhesions, and showed at necropsy some thickening of the meninges, especially in the frontal and occipital regions, very slight lymphocytic and plasma cell infiltration, and no disturbance in the cellular architectonics, so that unless one knew the previous history of the case no diagnosis of general paralysis would be possible from the pathologic picture. The authors think they are justified in concluding that with a remission of two and one-half years' duration and a stationary clinical picture there occurred also a regression of the pathologic findings. Two other cases showed a similar pathologic picture, though the clinical history showed the disease was not recent. In seven other cases the inflammatory phenomena, in comparison with those in untreated cases, were very slight. Of thirty-one cases with over five malarial attacks, more than one third showed a regression of the pathologic processes.

Anatomic remissions follow immediately on malarial attacks. This is shown by the fact that all patients who came to necropsy died after malarial treatment but had received no arsphenamine.

With the anatomic and clinical remissions occur changes in the spinal fluid findings, as pointed out by Kirschbaum and Kaltenbach, Pöltze, Gerstmann, Horu, et al. There follows a diminution in, and even disappearance of the usual reactions. The cell count is favorably influenced very early.

From a study of cases with death during the course of the malaria without remission, the authors show that the immediate effect of the malaria is to produce a great increase in the inflammatory process as shown by the marked

increase in lymphocytes and plasma cells. But while there is an increase in the inflammatory process, there is also a change in the character of the inflammation. Instead of the diffuse inflammatory process of general paralysis there occurs a specific, benign form of granuloma. The assumption that the malaria leads to augmentation in the inflammatory process finds support in the clinical symptoms, such as confusion, delirium, transitory hallucinations, etc.

ALPERS, Philadelphia.

OPTIC NEURITIS AND THE POSTERIOR NASAL SINUSES. L. BALDENWECK, Rev. d'oto-neuro-ocul. **2**:161 (March) 1924.

Seven cases of optic neuritis are classified in three groups: with sinusitis, frankly suppurative or recent; without actual sinusitis, but with mild abnormality of the nasal cavity; with nose and sinuses normal and no discoverable cause for the optic neuritis. In the first group were three cases: One was a case of bilateral chronic suppurative pansinusitis in which the ocular trouble that had existed for two years was almost immediately relieved after operation. In the second, the patient had complained for several weeks after an attack of coryza of a spot in the center of the right visual field. Mucopurulent secretion was seen on both sides of the (right?) middle turbinate, which soon disappeared. A central scotoma and a small hemorrhage in the region of the (right?) macula were found. Recovery with a pigmented zone occurred without operation. Baldenweck thinks that a timely operation in such cases would prevent sequelae. In the third, the patient had an acute multiple sinusitis which recovered; then there followed papilledema with peripapillary edema. The sphenoid sinuses were opened but no lesion was found. Recovery followed.

In the second group were two cases: The first patient, a hay-fever subject, rapidly lost vision in the right eye and in five months also in the left eye to the point at which reading became impossible. The right papilla and the temporal side of the left papilla were discolored. The middle turbinates were enlarged with polypoid degeneration on the right. Clouding of the right sphenoidal and fronto-ethmoidal regions was seen in the roentgenograms. The blood and spinal fluid Wassermann reactions were negative. Amputation of the right middle turbinate bone produced no amelioration of the eye condition. Another roentgenogram showed enlargement of the sella turcica. Vision rapidly improved after a pregnancy. The second was one of bilateral retrobulbar neuritis in which the patient had taken 1 Gm. of quinine daily over a long period. Central scotomas were present on both sides and marked hyperesthesia and rhinorrhea were found when the nose was swabbed. All other examinations gave negative results. Turbinectomy and exenteration of the left ethmo-sphenoidal sinus had no effect.

In the third group were two cases: acute right optic neuritis with normal nose and sinuses, enlargement of the thyroid gland and recurrent headache, which was followed by recovery; bilateral optic neuritis with only a slight hypertrophic rhinitis and no other discoverable cause, which was followed by optic atrophy.

The author concludes: 1. In cases of frank sinusitis — in those of chronic character operation should be performed; in acute cases operation is often indicated, and the ophthalmologist must decide the favorable moment. 2. In cases of frank or only probable meningo-encephalitic origin no operation should be performed because of the possibility of the presence of syphilis or multiple

sclerosis. 3. In cases of acute infectious retrobulbar neuritis operation should not be performed. 4. Cases of undetermined origin require most careful study before the question of operation can be decided.

DENNIS, Colorado Springs, Colo.

DAS KLEINHIRN: ANATOMIE, PHYSIOLOGIE UND ENTWICKLUNGSGESCHICHTE. R. BRUN, Schweiz. Arch. f. Neurol. u. Psychiat. **16**:183-197, 1925.

In this article Brun reviews some of the present conceptions of the anatomy, physiology and embryology of the cerebellum. The older anatomists, in discussing the cerebellum, concerned themselves with little more than sulci and lamellae. It was Burdach who coined the names of the various portions of the vermis. Flatau and Jakobsohn, in 1899, produced a work dealing with the comparative anatomy of the cerebellum of mammals. The recognition of the fissura prima dates from the work of Kuithan and Stroud, in 1895. The first great departure from the old descriptive anatomy was introduced by Elliot Smith, who attempted to homologize the various portions of the cerebellum in mammals, and introduced the transverse division of the cerebellum by the fissura prima and the fissura seconda. Bolk first attempted a functional division of the cerebellum. He found that, embryologically, the flocculus was the first part of the cerebellum to be well defined; this occurred at the end of the second month. At about the same time the fissura postnodularis of Smith put in its appearance. At the end of the third month the fissura prima was seen to begin in the midline. At the end of the fourth month the fissura secunda appeared just anterior to the uvula. At the end of the fourth month the lamellae appeared in the anterior lobe of the vermis.

Edinger and Comolli, in 1910, made another departure from the current classifications and in a sense returned to the oldest divisions of the cerebellum. They introduced the term paleocerebellum, which included the vermis and the flocculus, and the neocerebellum, which included the lateral hemispheres. This division is supported by phylogenetic and embryologic observations. The histogenic differentiation of these portions of the cerebellum is distinct. In the new-born child, the medullation of the paleocerebellum is almost complete, whereas that of the lateral lobes and the brachii is lacking. According to v. Valkenburg the first evidence of medullation occurs in the flocculus. Somewhat later, medullation takes place in the superior vermis, then in the median and posterior vermis, and finally in the neocerebellum. Biach, in 1910, found that the outer granular layer receded in the ninth month, but this occurred earlier in the vermis than in the lateral hemispheres. In animals that walk immediately after birth, medullation of the entire cerebellum is almost complete at birth.

Even pathologic observations support Edinger's conception. An elective atrophy which may be limited entirely to the neocerebellum has been observed a number of times. Indeed the neocerebellum usually suffers either in bilateral or unilateral degenerations of this character. The same thing is true in Marie's form of Friedreich's ataxia, and in olivopontocerebellar atrophy of Dejerine and Thomas.

WOLTMAN, Rochester, Minn.

CLINICAL FEATURES AND TREATMENT OF EPIDEMIC ENCEPHALITIS. KARL GROSS and MARINOS METAXAS, Jahrb. f. Psychiat. u. Neurol. **43**:63, 1924.

This paper is based on twelve cases of epidemic encephalitis, which are discussed from a clinical and therapeutic standpoint. There is nothing unusual

in the histories and clinical course of the disease that is not well known to American and English neurologists, so that the abstract of this paper will be limited to its therapeutic features.

The authors employed in their clinic the French method of administering arsenic. They prepared a solution of sodium cacodylate containing 90 mg. of arsenic to 1 c.c. of water. The injections were given every other day, the initial dose being 0.5 c.c. of the stock solution, which was increased by 0.25-0.5 c.c. per injection, the largest single dose given being 2 c.c. This dose was usually repeated twice, after which it was gradually decreased to the original dose. On an average a course of treatment consisted of fifteen injections with a total of 19.5 c.c. of the solution. In several cases a second and even a third series of injections was given, two weeks' rest being allowed between the two. The cases were not selected.

The results obtained, though favorable, were not as good as those reported by the French school. Some of the patients claimed that immediately after the injections they could notice improvement in the akinesia, tremor and rigidity. In those with ocular phenomena, only the spasms of the lids were considerably improved, whereas the ocular palsies and pupillary changes were only slightly improved. The injections were well tolerated. Only one patient showed evidences of arsenical poisoning, and these were transitory. Occasionally the injections were followed by an increase in salivation and in the excretion of urine; in other cases, the opposite of this was observed. Many of the patients, in contrast to the experiences of the French, complained of a disagreeable odor of garlic from the arsenic. None of the patients showed an increase of weight during or after treatment. In one case the arsenical treatment was preceded by treatment with malaria and in another by treatment with typhoid vaccine. The severity of these cases was apparently unaffected by any of these measures. Two of the patients were subjected during the arsenical treatment to a course of systematic active and passive movements. This therapeutic aid is recommended for its favorable psychic effect — transitory as it was. The authors state that owing to the short period that these patients were under observation, they are not in a position to give a final and definite opinion as to the efficacy of such large doses of cacodylate of soda. They believe, however, that in view of the sad outlook that this disease presents from a prognostic standpoint, and in view of the comparative harmlessness of the method, it deserves further trial.

KESCHNER, New York.

CEREBELLAR AND PYRAMIDAL REACTIONS IN THE COURSE OF AN EXTRADURAL ABSCESS OF OTITIC ORIGIN. J. A. BARRÉ, Rev. d'oto-neuro-ocul. **1**:81 (Feb.) 1923.

The case described was one of acute left mastoiditis with a large extradural abscess. The dura was exposed over a large area in the posterior fossa and a smaller one in the middle fossa, including the portion over the point of the semilunar lobe and part of the digastric lobe in front of the sigmoid sinus, the sinus itself and a small part of the inferior border of the occipital lobe.

The neurologic examination revealed: psychic state normal; reflexes normal, except that there was a marked diminution of almost all the skin reflexes on the right side; some mild evidences of cerebellar disturbances in the left limbs. The only positive evidences of this last were from tests of passivity and dysmetria in the lower limbs. The voluntary and reflex motility was slightly disturbed. The eyegrounds were normal; the spinal fluid was normal, and

there was no fever and no vomiting. The labyrinth examination revealed no nystagmus on looking straight or in convergence, but a few fine twitches on looking laterally. The extended arms deviated to the left. The Romberg test was negative.

When a cerebellar lesion is suspected in an ear case, one should not always expect to find a complete cerebellar syndrome. A few mild signs, if clear and positive, are sufficient. The examination of the cerebellum should always be followed by inquiry into the psychic state, the condition of the cerebral cortex, the intracranial tension and the spinal fluid.

In the later studies, chilling experiments were made on the exposed cerebellar dura. A jet of ethyl chloride was sprayed for fifteen or twenty seconds over the semilunar lobe, the patient being seated upright. This should paralyze the outward pointing center for the left arm (Bárány) and cause a movement of the arm to the right. There was no modification of the position of the arm. Ethyl chloride was sprayed on the same place for almost a minute. After thirty seconds' spraying the patient felt badly; a marked rotary nystagmus to the right occurred, and both arms deviated to the left (typical labyrinth reaction). All phenomena lasted about two minutes. Some minutes afterward the extended arms showed no lateral deviation, but a slight lowering of the left arm occurred.

Irrigation of the left ear with 15 cc. of water at 25 C. produced no reaction. Irrigation of the right ear with 75 cc. of water at 47 C. produced a nystagmus to the right, and both arms deviated 20 cm. to the left. There was no vertigo.

Five days later, heat (45 and 50 C.) was applied to the same place. At the end of one minute and forty seconds, fine twitches of nystagmus occurred to the left, and the arms deviated to the right. When the eyes closed, the nystagmus increased and lasted for several minutes, during which time no hypermetria, no adiadokokinesis of the left arm and no anomaly in the left heel-to-knee test were observed.

DENNIS, Colorado Springs, Colo.

THE CENTRAL TRACTS OF THE NERVUS VESTIBULARIS. ISAAC H. JONES and WILLIAM G. SPILLER, Brain **48**:334 (Sept.) 1925.

A boy, aged 15, who for over a year, had suffered from epidemic encephalitis, consisting chiefly of respiratory difficulty, profuse sweating spells, convulsions and incoordination and disturbance of equilibrium in walking, presented the following features on examination: the general picture was one of a mild parkinsonian syndrome in which coordination was fairly accurate but the spontaneity of the voluntary movements was impaired; the pupils were equal and reacted well to light; movement of the eyeballs upward and convergence were greatly impaired; there was a persistent spontaneous nystagmus; on looking upward there was vertical nystagmus, while to the right and left it was of mixed horizontal and vertical type; on looking downward there was no nystagmus; there was perfect auditory function in both ears, but complete absence of vestibular function in both. The rotation and caloric tests failed to produce nystagmus, vertigo, past pointing, falling, pallor, sweating or nausea. There was, therefore, a complete vestibular block, the location of which appeared to be within the brain stem shortly after the entrance of the eighth nerves. The intellect was at all times very keen.

Necropsy findings revealed no gross changes in the brain. The entire brain stem was studied histologically by serial sections with the following interesting results: At the exit of the nervus vagus and nervus hypoglosus there

was a limited area of degeneration confined to the tegmentum and situated on each side of, but not implicating, the raphe. As these two areas of degeneration were followed upward they gradually approximated, until at a level of a little below the vesticular and facial nerves they had united into one central area; the raphe at this point was degenerated. This picture persisted up to the lower part of the pons. The degenerated area extended across the central part of the medulla oblongata well posterior to each nucleus olivaris, so that the striae of Held and the corpus trapezoides were not involved. At no level were the radicular fibers of the nervus cochlearis or nervus vestibularis or their terminal nuclei implicated by the degenerated area. The nucleus ambiguus showed evidence of encephalitis and there were distinct nerve cell changes in the triangular and Deiter's nuclei. In the middle of the pons were more diffuse encephalitic changes, which became very pronounced in the midbrain.

A thorough review of the central connections of this complex nerve is given, the work of C. Winkler being extensively quoted. Dr. Spiller concludes from this study that all or nearly all of the central tracts concerned with vestibular function pass through the area represented by this degeneration, and that a few fibers of the cochlear system are represented in this region, the majority passing ventral and dorsal to this area. The degeneration, therefore, involves only the central tracts of the vestibular system.

STACK, Milwaukee.

PHOTORECEPTORS OF LUMBRICUS TERRESTRIS, WITH SPECIAL REFERENCE TO THEIR DISTRIBUTION, STRUCTURE AND FUNCTION. WALTER N. HESS, J. Morphol. **41**:63 (Dec. 5) 1925.

A special type of cell, called by Hesse "Lichtzelle," was found in the photosensitive and was absent from the nonphotosensitive regions of *Lumbricus terrestris*. These photoreceptor cells are found in the epidermis of all body segments, as well as in the body cavity, and in certain nerve enlargements of the prostomium and of the last segment. So far as the epidermis is concerned, the prostomium is most abundantly supplied with these cells. The caudal segment contains about as many as segment 2. Those segments near the middle region of the body have very few. The intersegmental and ventral regions of the different segments, except certain of the distal ones, are apparently barren of such cells. Wherever the epidermis of the earthworm is most sensitive to light the number of photoreceptors is greatest. These cells, in addition to being supplied with large nerves, contain a cylindric cytoplasmic structure, the optic organelle called by Hesse the "Binnenkörper." The inner portion of the optic organelle is composed of a transparent hyaline substance, the lens, which is surrounded by a neurofibrillar net, the retinella, formed from fibrils which enter the cell from a large nerve. These cells, when placed in hanging drops, focus light on the outer border of the optic organelle — that is, on the retinella — irrespective of the direction from which the light enters. The structure of the photoreceptor cells, together with their refractive behavior, seems to indicate that the nerve fibers of the retinella are the direct receptors of photic stimuli. The photoreceptors are apparently specialized epidermal cells. Those in the nerve enlargements of the prostomium, as well as those of the caudal segment, are probably cells that have wandered from the epidermis into the interior of the body along the nerves. The reactions of the worm aided by the photoreceptors are such as to keep the animal within its burrow except during darkness when the chief enemies of the worms are usually inactive. Pigment is not so associated with the photoreceptors as to indicate

any photochemical relation to their stimulation. It is suggested that there is probably direct stimulation of the nerve fibrillae of the retinella by light, which may be electric in nature.

A comparison of these cells with the visual cells of leeches shows that the two contain structures that are similar, both in structure and in function, and as they are probably similar in origin, they may be regarded as homologous. It is apparent that the condition of the photoreceptors in *Lumbricus terrestris* is intermediate between that in primitive oligochete annelids with only epidermal photoreceptors, and that in those forms which have paired anterior eyes.

WYMAN, Boston.

PAPILLARY STASIS AND DECOMPRESSION. VELTER, Rev. d'oto-neuro-ocul. **1**:93 (Feb.) 1923.

Velter discusses the subject from the ophthalmic standpoint. He recalls the fact that, in its beginning, papillary stasis can be recognized only by ophthalmoscopic signs; vision may remain intact for a long time; in a suspected case of hypertension the eyegrounds should be examined frequently because stasis is often evolved in successive, increasing steps, and may at times gravely compromise vision even in a few hours. Transient complete blindness is an alarming sign, each attack leaving increased lowering of vision.

Two methods of decompression are lumbar puncture and craniotomy, each having its own indications. One should always begin with lumbar puncture. If its action is favorable it should be prudently repeated, but the ease of performance and repetition is its greatest danger, since one should not delay radical decompression too long, from eight to ten days at most. The ophthalmologist should decide the time for operation with all the energy and authority possible, as prolonged hesitation will end in disaster. Mercurial treatment should always be given. Decompression should be done rapidly and without search for the lesion unless a precise localization has been made. Usually the subtemporal or temporoparietal approach is best. The dura should not be incised unless a tumor is discovered at once, and then not at the primary operation. The trephine opening should be at least 6 or 8 cm. in diameter and the bone should not be put back in place.

The results are good and the operation without danger. In twenty-seven cases there were seven excellent and durable results; eleven showed improvement, seven transitory benefit and two no improvement. If the vision is notably diminished, the operation maintains it without further loss. Vision begins to show improvement before the eyegrounds, and it is often several weeks or months before the nerve head becomes normal. Even in favorable cases, some traces, such as tortuous veins, pigmentation and irregularity of the papilla, remain. Since the destruction of vision may progress very rapidly, decompression should be done early.

The etiology is important in prognosis. In tumor cases the operation does not modify the growth of the tumor, but in cases of serous meningitis or ependymitis, recovery may be permanent. One patient, clinically cured with recovery of perfect vision for two years, had extensive glioma of one hemisphere; another, likewise cured, died after two years of pulmonary tuberculosis. A tuberculous cicatrix was found in the cerebellum. Recovery from hypertension does not preclude the presence of a tumor. There is no such thing as pseudotumor; either there is a tumor or there is not. It is often impossible to know the exact cause of hypertension.

DENNIS, Colorado Springs, Colo

THE FUNCTION OF PERIPHERAL NEURONES IN THE CONDUCTION OF IMPULSES IN THE SYMPATHETIC NERVOUS SYSTEM. A. QUERIDO, Am. J. Physiol. **70**:29 (Sept.) 1924.

Querido reports observations on the nature of autonomic impulse transmission in the nictitating membrane of the cat. This tissue he regards as a smooth muscle system ideally suited for study because of the simplicity of its structure and innervation. It receives innervation from only one autonomic center, the superior cervical ganglion, and contains only postganglionic fibers, no ganglionic cells and no antagonist system. In the experimental animals the superior cervical ganglion was exposed, and induction shocks were applied to the preganglionic and postganglionic trunks. The response contractions of the membrane were recorded on a rotating drum. Inhibition of muscular contraction may be induced by stimulation of the postganglionic elements, whereas no apparent effect of this nature was found from preganglionic stimulation, even with shocks of considerably higher frequency. Maximal contractions resulted from preganglionic excitation with shock rates of lesser magnitude than was required for maximal contraction in postganglionic excitation. Querido is, therefore, led to conclude that impulses of widely different frequency, applied preganglionically, develop impulses in the ganglion which proceed distad at optimum frequency. This optimum frequency represents a range of from 120 to 160 per second. It appears also that with postganglionic excitation, the latent period varies inversely with the magnitude of the stimulus, whereas the latent period seems constant for preganglionic excitation, whether maximal or submaximal.

From these observations one is led to conclude that in the passage of centrifugal impulses through sympathetic ganglia, a transformation to optimal occurs for both frequency and intensity. That is, the ganglion cells of this system seem to possess a capacity for adapting impinging stimuli to the special nature and requirements of the tissue to which they are directed. The transforming of rhythm, Querido explains, is effected by the setting up of an intrinsic rhythm within the ganglion cells and the transformation of intensity as a manifestation probably of the action of the law of "all or none."

RAPHAEL, Ann Arbor, Mich.

THE RELATION OF TRANSPLANTED EYES TO DEVELOPING NERVE CENTERS. RAOUL M. MAY and S. R. DETWILER, J. Exper. Zool. **43**:83 (Nov. 5) 1925.

Homoplastic grafts of the eyes were made on embryos of *Amblystoma punctatum* in the tail-bud stage. The optic vesicle and superjacent lens placode were transplanted to the otic region of a second embryo from which the auditory vesicle had been previously removed. Essentially normal eyes developed. An optic nerve developed in all cases studied (fifteen cases). The olfactory placode was incidentally transplanted in part and developed, in all cases, to a varying extent. An olfactory nerve developed in thirteen of the fifteen cases. The eye in some cases was partially distorted. This distortion occurred especially at the place at which the optic nerve made exit from the eye to form a connection with nerve centers. Fifteen larvae were examined. The transplanted optic nerve in four cases terminated at the pigment layer of the eye, in three cases it terminated in the mesenchyme, in three cases it

connected with the unremoved portion of the acousticofacial ganglion, in four cases it connected with the glossopharyngeal-vagus ganglionic complex, and in one case it connected with the medulla. The transplanted olfactory nerve terminated in the mesenchyme in five cases; it connected with the gasserian ganglion in two cases, with the facial portion of the seventh-eighth ganglion in three cases and with the ninth-tenth ganglionic complex in three cases. Neither rotation of the eye nor the amount of cartilage which develops about it are critical factors in determining the connections of the optic nerve. Removal of the auditory vesicle brings about a cellular reduction of 60 per cent in the seventh-eighth ganglionic mass. There was a hyperplasia of the ninth-tenth ganglionic complex with which the optic nerve connected, and these ganglia shifted cephalad toward the entering optic nerve. The ingrowing axons from hyperplastic ganglia caused no hyperplasia in the medulla. Direct connection of the optic nerve with the medulla brought about a cellular hyperplasia and a shifting of the cells from the gray matter into the white toward the point of entrance of the nerve. The hyperplasia produced in the nerve centers varied from 22 to 52 per cent of the normal.

WYMAN, Boston.

CRITIQUE OF THE THEORY OF MENTAL RECAPITULATION. I. D. SUTTIE, J. Neurol. & Psychopath. **5**:1 (May) 1924.

The author thinks it can be shown that the theory of mental recapitulation has outlived its usefulness, that much that has been promised on its behalf can never be fulfilled, and that its application to psychology can be of no conceivable value to that science. Biologic opinion is not united as to the adequacy of the evidence for recapitulation or even as to the necessity of this interpretation of the ontophylogenetic parallelism. As ancestral and infantile environments differ and consequently lead to a divergence between ontogeny and phylogeny in regard to special adaptive phases and characters, therefore mind, the most plastic and adaptive of all characters, should be the least likely to recapitulate. The germ-plasm must be the mechanism which maintains the parallelism between development and evolution.

Biologic theory implies that the recapitulatory tendencies come within the sphere of that developmental factor we call heredity. So far as development is molded by the environmental or "nurtural" factor, biologic recapitulation cannot occur. The most important achievement of the psychanalytic movement has been to show the pathogenic effect of certain emotional relationships in childhood. Its unique contribution is the canon of psychic determinism, which asserts that the causes of acts and thoughts should be sought in the antecedent experience of the individual. It is to be hoped that the exponents of psychanalysis will have further success in relating mental characteristics and abnormalities to factors and abnormalities of up-bringing, thus adding to our prophylactic, therapeutic and educative resources. In claiming that biologic recapitulation holds in the sphere of mental development, they are admitting a restriction in the applicability of their method. It would be sound method for psychology to scrutinize critically any hypothesis assigning to nature as against nurture a preponderant rôle in mental development. The more purely logical and metaphysical arguments for mental recapitulation break down in many places. We may conclude that mental development is more closely related to culture evolution than to brain evolution.

FAVILL, Chicago.

IDENTIFICATION OF THE CELLS AND FIBERS CONCERNED IN THE INNERVATION OF THE TEETH. WILLIAM F. ALLEN, J. Comp. Neurol. **39**:325-343 (Dec. 15) 1925.

In two Nissl series through the semilunar ganglia from dogs, in which all of the teeth but the most median incisors had been extracted from both left jaws, with very little laceration to the gums or alveolar processes, it was shown that about one eighth of the total number of cells exhibited chromatolysis or Nissl degeneration. Of the total number of altered cells about three fourths are large and medium sized cells and only one fourth are small cells. These altered cells appear to be scattered uniformly throughout the entire ganglion and not confined to a triangular mass at the base of the maxillary and mandibular trunks, as described by Perna for the monkey. A study of modified Ramón y Cajal silver preparations of the main branches of the nervus trigeminus discloses that certain areas of the section in which the small fibers are more abundant contain a small proportion more of small nonmedullated or sparsely medullated fibers than they do of large and medium-sized medullated fibers. In other parts of the same section the reverse would be true. Both the nervus alveolaris inferior and the cephalic end of the nervus maxillaris are not characterized by any great predominance of small fibers over large and medium-sized fibers. They contain no more small fibers than the other branches of the nervus trigeminus that were studied.

Commenting on these and other observations the author offers the following tentative hypothesis: The size of the cell body (trophic center), the size of the axon, and possibly the size of the myelin sheath (if a nutrient center), are dependent primarily on the load that the neuron is, normally and under special conditions, required to propagate. This may take into account the size and importance of the terminal ending, the length of the axon and the amount of terminal branching of the neurites or dendrites. There may be some difference in the structure of a neuron that is continually being called on to deliver a weak impulse and one that is occasionally required to propagate a very strong impulse.

HERRICK, Chicago.

STUDIES ON THE NERVOUS REGULATION OF PROGRESSION IN MAMMALS. N. B. LAUGHTON, Am. J. Physiol. **70**:358 (Oct.) 1924.

Laughton finds that serious interference with normally coordinated progression results unless, in the case of dogs and cats, the caudal two-thirds of the thalamus is left intact and, in the case of rabbits, the cephalic two-thirds of the pons. Coordination in progression is destroyed also as a result of median longitudinal section through these regions, indicating the importance of the integration factor for the mediation of this mechanism. Definite anatomic centers of control as such, however, were not determined, coordination control being probably dependent, in a general way, on the orderly integrated function of the brain stem complex as a whole.

For the rabbit, it was noted that the cerebellum is not essential for properly coordinated progression movements, but, in decapitated animals, such movements were found to be entirely abolished. For all the animal types utilized, even the marked rigidity consequent on decerebration appeared to exert no special inhibitory influence on progression control. It was determined further that whereas, in rabbits, alternate rhythmic movements constituting the simple reflex hind leg step could be effected through the lumbosacral cord, no response approximating the synchronous bilateral movements of normal progression

could be elicited from this region alone. A finding of interest also was the observation that decerebrate shock following thalamic section was most marked in rabbits, less in cats and least in dogs, indicating, as Laughton suggests, the possibility of the implication of the phylogenetic factor with reference to grade or degree for this type of shock.

Finally, although kittens and pups reacted to decerebration at similar levels with very little shock and rigidity as compared with the adult animals, a similar effect was induced on progression control. This leads the author to conclude that in all probability shock and rigidity cannot be considered as in any sense primary to the interferences described respecting the mechanism of progression regulation.

RAPHAEL, Ann Arbor, Mich.

SENSORY OCULAR DISTURBANCES IN ENCEPHALITIS LETHARGICA. AUBINEAU and GRENIER, Rev. d'oto-neuro-ocul. **2**:186 (March) 1924.

Visual complications in epidemic encephalitis are rare, but transitory amblyopias have been reported, as well as quite a few objective lesions of the eyeground accompanied by alterations of vision. The authors arrange the cases in two groups: (1) with congestion of the nerve head and papillary stasis from increased endocranial pressure; (2) with little or no ophthalmoscopic change, but with serious functional trouble from affection of the optic nerve (central scotoma, alterations of the visual fields) and visual disturbances from affection of the cortex or the superior optic pathways.

Two cases are reported, in both of which the etiology was clear and the spinal Wassermann reaction negative. In the first case the eye findings were: trembling of the lids; reaction of the pupils to light but not in accommodation; visual acuity diminished and variable; central scotoma in which white, colors and movement of the hand were not perceived. In the second case, pupillary reactions were present but diminished for light and in accommodation; the pupils were unequal; a divergent strabismus was present; there were no eyeground changes, and trembling of the lids was present. Parkinsonian symptoms were marked in the face. Vision: right eye, 2/10, left eye, 1/10. There was no central scotoma, but peripheral retraction of the visual fields was present.

The first case showed an absolute and extended scotoma which was referred to a retrobulbar neuritis. The location of the lesion in the second case was not determined. In the first case the amblyopia occurred early in the encephalitis; in the second, the sensory troubles developed late.

While most of the histologic changes in encephalitis are found in the mesencephalon, the congestion affects the whole encephalon; any part, including the meninges, may be affected; inflammatory lesions have likewise been found at the level of the cranial nerves. This explains why visual function may be compromised.

DENNIS, Colorado Springs, Colo.

STUDIES IN GASTRIC SECRETION. I. THE PSYCHIC SECRETION OF GASTRIC JUICE UNDER HYPNOSIS. A. B. LUCKHARDT and R. L. JOHNSTON, Am. J. Physiol. **70**:174 (Sept.) 1924. II. GASTRIC SECRETION IN SLEEP. R. L. JOHNSTON and H. WASHEIM, Am. J. Physiol. **70**:247 (Oct.) 1924.

In the first paper, observations were made on a male subject, aged 21. Gastric samples were collected at ten minute intervals prior to the giving or suggesting of meals, and at fifteen minute intervals after them. The material

obtained was analyzed for free and total acidity. The authors conclude that the gastric glands respond more promptly to the test meal (Ewald) under hypnosis than in the unhypnotized condition. Likewise it was found that, in hypnosis, the suggested meal is followed by a secretion curve of as high an acidity as occurs in response to an actually ingested test meal. A secretion curve was also noted during the establishment of the hypnosis status. This curve subsided in the course of from two to four hours and was due probably to the removal or diminution of the inhibitory influence of higher centers normally present in the waking state.

In the study of gastric secretion during sleep, a single adult man was also used; in general, essentially the same technic was employed as in the hypnosis experiments. This investigation showed that preparation for sleep seems to induce an increased acidity which lasts until at least an hour after sleep has become established, and reaches its acme at the point of greatest sleep intensity. Sleep following the ingestion of a test meal seems to cause delay in gastric emptying and increased acidity, with marked delay in the rate of fall as compared with that observed in sleep which has not been preceded by the administration of a meal. It was found also that the curve of secretion, in the absence of test-meal administration, is practically identical in sleep and hypnosis. Likewise it was determined that the amount of gastric juice secreted per hour was more than twice as much for waking states at night as in sleep.

RAPHAEL, Ann Arbor, Mich.

ACUTE MULTIPLE SINUSITIS: RECOVERY. PAPILLEDEMA, WITH PERIPAPILLARY CONGESTION. OPERATION: NO PERCEPTIBLE LESION OF THE SPHENOID SINUS. A. CANTONNET, E. MARTERET and L. BALDENWECK, Rev. d'oto-neuro-ocul. **2**:105 (Feb.) 1924.

A man, aged 30, had suffered an acute left frontomaxillary sinusitis five months previously. Recovery was complete, although slight clouding of the left frontal and maxillary sinuses was seen in roentgenograms. Following a plunge, feet first, into a pool, frontal headache developed and lasted two or three days. A few days later, he noticed visual difficulty. Examination of the left eye showed indistinct outline and reddening of the papilla; small arteries, veins tortuous and dilated; bloody suffusion below the papilla. Vision was 9/10. The nasal cavities were normal. Other examinations revealed no abnormality. The Wassermann reaction was negative.

When the sphenoidal cavity was opened by the transeptal route, the cavities were found to be small and normal. Gradual restoration of vision and return of the eyeground to normal occurred.

The authors discuss two hypotheses: reinfection of sinuses, and mechanical disturbance of the ocular circulation from compression of air in the nasal cavities by the force of the dive. They dismiss the first. Reasoning from the fact of air emboli following antrum puncture and inflation, they think there may have been in this case a slight trauma of the mucous membrane, followed by air penetration into the vessels, which reached eventually the ocular vessels; following this, vacuum sinusitis occurred in the sphenoidal sinus due to a valve-like action of tissue around the opening, which maintained a disturbance of the ocular circulation until the opening of the sphenoid relieved the condition.

DENNIS, Colorado Springs, Colo.

THE PARKINSONIAN SYNDROME WITH TUMORS OF THE INFUNDIBULO-HYPOPHYSIAL REGION. ORZECHOWSKI and W. MITKUS, Rev. neurol. **2**:1 (July) 1925.

Some tumors of the infundibulo-hypophysial region penetrate far into the cavity of the third ventricle reaching the roof and determining almost always dilatation of all the ventricles. This is especially true of tumors developed from the Rathke pouch, which frequently contain calcareous deposits. From the clinical point of view, this type of tumor elicits a hypophysial syndrome and, in addition: extrapyramidal symptoms, isolated or grouped in a parkinson-like syndrome, with marked involvement of the lower extremities; a partial thalamic syndrome, characterized by hyperesthesia to pain and sometimes by spontaneous pain in the body and extremities; a marked tendency to somnolence which is more marked than in the simple hypophysial tumors; very often obnubilation, complicated sometimes by an amnesic syndrome; slight cerebellar symptoms; marked dilatation of the ventricles as shown by air insufflation and calcareous spots in the roentgenogram. Many of the symptoms have already been described by Weisenburg in his study of tumors of the third ventricle, but the writer insists particularly on roentgenologic studies, and studies on the hydrocephalus and on the extrapyramidal and the thalamic syndrome.

FERRARO, Washington, D. C.

STUDIES ON THE PERMEABILITY OF THE MENINGES. II. THE PERMEABILITY IN SYPHILITIC DISEASES. F. K. WALTER, Ztschr. f. ges. Neurol. u. Psychiat. **97**:192 (June) 1925.

1. The permeability quotient in normal persons is a constant quantity.

2. In general paralysis the permeability of the meninges is definitely increased in a high percentage of cases.

3. In many cases the permeability relations are definitely connected with the clinical picture.

4. Syphilis without neurologic findings and without changes in the fluid causes no change in the permeability.

5. Syphilitic endarteritis causes no change in the permeability.

6. Malarial treatment of general paralysis has a definite influence on the permeability relations.

ALPERS, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY WITH THE SECTION OF NEUROLOGY AND PSYCHIATRY OF THE ACADEMY OF MEDICINE

Regular Meeting, Nov. 10, 1925

I. Abrahamson, M.D., *President, in the Chair*

The Abortive Type of Friedreich's Ataxia. Dr. Charles R. Rombold (by invitation) and Dr. Henry Alsop Riley.

This article will be published in full in the Archives.

The Neuroses in the Course of Chronic Epidemic Encephalitis. Dr. Foster Kennedy.

Heretofore, descriptions of mental illness have been but descriptions of mental mechanisms, with no regard whatever to disordered physical states of neural apparatus. Cases of encephalitis have been described in which altered emotional tone was the chief result of the disease, accompanied by altered vital rhythm, as in sleep, respiration or the intake and output of fluid; cases too in which abulia was the outstanding feature. The identity between so-called "functional" illnesses, such as the neuroses, and many of the post-encephalitic sequelae must be emphasized. These latter have been ascertained to arise from peri-aqueductal lesions. Must the cause of the former be searched for solely in psychic terms? Is it not necessary to cease being satisfied with a purely mental explanation due to environmental or instinctive stress? Such explanations are useful as far as they go, but so far are they from being an ultimate analysis, "they are but figure-skating on the surface of the problem"; they have served to make profitable the time which must pass while physiology takes on a larger stature and more definite form, but the neurologist of the future will try by the examination of defects of structure to illuminate defects and perversions of mental function and emotional personality.

The Mental Symptoms in Epidemic Encephalitis. Dr. R. T. Folsom (by invitation).

The basis of this study consists of an analysis of the case histories of seventy-one patients admitted to the Manhattan State Hospital and diagnosed as psychoses with epidemic encephalitis. The first patient was admitted April 2, 1920, and the last July 16, 1925. There were approximately twice as many males as females, the numbers being forty-eight and twenty-three, respectively. The number admitted each calendar year is of interest as a possible reflection of the incidence of encephalitis in the general community. The admission curve was high in 1921, then dropped somewhat for three years, and has reached its peak with twenty-one cases in the year 1925.

The age incidence in this series showed that the most vulnerable period was the second decade which contained thirty-six cases, and this vulnerability held good for each sex. In the entire series there were twenty children under

14 years of age, fifteen boys and five girls. In this series the greater age incidence occurred in the period of preadolescence and adolescence, which may have some correlation with psychiatric aspects to be mentioned later.

The incidence of influenza prior to the onset of encephalitis was inquired into, and a positive history of influenza was found in twenty-four of the total of seventy-one cases. It was also found that fifty-nine had suffered from lethargy and nine from inability to sleep, and a history covering sleep disturbance was lacking in three.

In every case of this series, neurologic syndromes of the types frequently described in encephalitis were present. Some of the syndromes were: nine parkinsonian ocular; eight oculofacial; seven uncomplicated parkinsonism; seven parkinsonian myoclonic, and thirty-seven other neurologic admixtures containing four cases or less each.

A study of the personality make-up was made for the purpose of determining whether there was any correlation between the type of make-up normal to the patient prior to the onset of encephalitis and the psychotic symptoms exhibited. The compilation showed that forty-one were of a normal make-up; sixteen of an unstable make-up; thirteen of a seclusive make-up, and one of a make-up unknown. Of the forty-one of normal make-up, thirty-five exhibited psychoses consisting of emotional instability, and six had deliria. Of the sixteen unstable individuals, fourteen had a psychosis consisting of emotional instability, one had a paranoid trend with an acute episode of apprehensive agitation. Of the thirteen of seclusive type, eight had an emotional instability, two had delirium, one had delirium followed by emotional instability, one had an acute paranoid hallucinatory episode, and one had a dementia praecox syndrome. These figures would seem to indicate that irrespective of the type of make-up a considerable majority of each type of person exhibited a clinical picture characterized by pathologic emotional instability, the outstanding exception to this being a seclusive individual in whom a dementia praecox psychosis ran true to the make-up.

A consideration of the psychiatric clinical picture presented in this material permits a division into four groups: sixty-three cases characterized essentially by emotional instability; six cases of uncomplicated deliria; one case consisting of an acute hallucinatory paranoid episode, and one dementia praecox syndrome. The emotional instability group of sixty-three patients comprises such an overwhelmingly large number of the cases that one is led to consider it the paradigm of psychoses associated with encephalitis. Of this group of sixty-three cases, fifty were of uncomplicated emotional instabilities; there was one case of paranoid trend followed by emotional instability; three were complicated by apparent hysterical manifestations, and three were simple depressions; six had a delirium followed by emotional instability. It is to be noted that the delirious cases mentioned refer to the psychotic condition presenting itself in the hospital and do not include the history of delirium which is so often present in the earlier encephalitis.

In this large group of emotional instability cases, the history obtained in the great majority is that of a person who, following the attack of encephalitis, became irritable and combative, and indulged in screaming or crying episodes and fits of temper, and theft and sex delinquencies. Under hospital regimen the instability tends to become more episodic, perhaps as a result of disciplinary measures; and during the quiet intervals the patients have expressed an unusual degree of insight into their abnormal behavior and lack of emotional control. These cases are further characterized by clear sensorium, lack of memory impairment and an absence of hallucinations and trends.

The psychotic manifestations shown by the twenty children under 14 years of age were, in general, characterized by an extraordinary degree of irritability, impulsiveness, incorrigibility and theft and sex delinquencies. The emotional instability in adults was less intense in its manifestations; the theft and sex delinquencies did not prevail in the older patients, with a few exceptions in later adolescence. The simple depressions occurred in the fourth decade; the deliria, with two exceptions, occurred among the older patients in the third, fourth and fifth decades, admitted a comparatively brief time after the onset of encephalitis. On the other hand, the date of onset of encephalitis prior to admission in the emotional instability cases was measured usually by years instead of months.

The ultimate course of the cases is of some interest. Ten patients were transferred to other hospitals, three improved and seven unimproved at the time; their present condition is unknown. Six patients died in the hospital, the causes of death being as follows: one, pulmonary tuberculosis; one, terminal exhaustion; two, myocarditis; one, chronic endocarditis and one, bronchopneumonia. In all these cases necropsy permits were refused. Twenty-three have been discharged from parole, of whom six had recovered, eleven had improved and six were unimproved. None of these patients received any sodium iodide in treatment. Five are on parole at present; of these, four are much improved, and one is improved. Three of these had received injections of sodium iodide, and two had had no special medical treatment. Twenty-six patients remain in the hospital; of these, ten have received treatment consisting of intravenous injections of sodium iodide, 50 cc. of a 10 per cent solution twice a week, and are improved; four are much improved under the same treatment; ten are in a stationary condition, of whom three have received no treatment, six have received treatment only occasionally because of refusal to submit to intravenous injections, and three have received sodium nucleinate subcutaneously because of veins too small for intravenous treatment.

It should be noted that with the exception of the one case cited none of this series of patients still remaining in the hospital shows evidence of a dementia praecox psychosis. A comparison of patients discharged from the hospital before any special medical therapy was given would indicate a tendency to spontaneous remissions in a certain number of cases. On the other hand, intravenous treatment with sodium iodide, which was commenced in November, 1924, and which at present is in only a tentative stage, would seem to indicate that cases do better under such therapy than without it.

UNUSUAL SIGNS OF EPIDEMIC ENCEPHALITIS. DR. L. GRIMBERG.

Three cases of an unusual postencephalitic syndrome and unusual development, all manifested a torticollis with myoclonic seizures, pes equinus, lateral curvature of the vertebral column and a peculiar gait. One patient showed a slight rigidity of the right side of the body with slight hypotonia of the left side. Another patient had a typical decerebrate-like attitude during the myoclonic seizures, and the third patient showed marked hypotonia of the extremities, except the right arm, which was slightly rigid. The third case began four years before as a typical parkinsonian syndrome following encephalitis; the present state, that of hypotonia, developed gradually. The present state is a dystonia, but with absence of dystonic movements.

Analysis of the posture and movements in the three cases classes them as belonging to the syndrome of dystonia, an unusual form of post-encephalitic picture.

DISCUSSION

DR. J. RAMSAY HUNT: Epidemic encephalitis has been so frequently the subject of discussion in the past few years that the usual symptoms and clinical features are familiar. To my mind, the most important points for emphasis are the peculiar nature of the sequelae and the marked tendency to recurrences manifested by this disorder. In this respect its behavior is very different from acute poliomyelitis, also an inflammatory infection of the central nervous system. In poliomyelitis there are paralytic residuals depending on the severity of the attack, but no recurrences. In epidemic encephalitis, however, the tendency to recurrence is marked, resembling in this respect multiple sclerosis and syphilis of the central nervous system. It is now generally recognized that parkinsonism occurs in about 25 per cent of the cases, although this percentage has varied somewhat in different epidemics and in different countries. Von Economo, who first called attention to this disorder in 1917-1918, also recognized the existence of a chronic recurrent type. In a histologic study of such a chronic case he found, as one would expect, the coexistence side by side of recent inflammatory and older chronic lesions. Greenfield and Buzzard also found chronic changes in the older cases, and laid special emphasis on the calcareous deposits and degeneration. As I look back on my experience with this disease I can recall a few cases of special interest from the standpoint of symptomatology. During the later period of an epidemic, I saw a boy, aged 12, who had taken cold, with a moderate elevation of temperature. The following day slight choreiform twitchings developed which lasted forty-eight hours only. In ten days he was well with the exception of insomnia which lasted for months. There was a complete reversal of the usual sleep mechanism. He was drowsy during the day, and had a curious wakefulness with restlessness coming on rather punctually at night. Before the epidemic I do not know what we would have called this. We might have regarded it as a very mild or abortive type of Sydenham's chorea. Neurologists were quick to see this analogy in regard to cases of Sydenham's chorea, which has certain infectious qualities; Marie and Foix examined a fatal case and found lesions similar to those seen in encephalitis in the central portion of the brain, especially around the corpus striatum.

I recall another case of a woman who had paresthesias which seemed to be of an organic type and a typical Argyll-Robertson pupil. There were no other symptoms of tabes. The reflexes were normal. The neurologic and serologic findings were entirely negative. The past history showed that, two years before, the woman had had what was regarded as a mild attack of "grippe." She was a little delirious for a night or two, and that was all. The assumption is that the attack was one of encephalitis, comparatively slight, and that the later symptoms were residuals referable to the midbrain and the thalamic region.

The mental aberration and changes in character one sees, especially in children, are very interesting. The explanation of some of these peculiar reactions should be looked for in the personality of the individual; when the brain is affected by organic and toxic lesions, paralysis and inhibitions result, which release a symptomatology that will vary with the peculiarity of the person.

DR. GEORGE H. KIRBY: Dr. Folsom's report on the cases observed at the Manhattan State Hospital is a welcome contribution because it throws some light on the important question of what form the late and residual mental reactions have taken in epidemic encephalitis. It is well known that in the

initial or acute phases of the disease, presumably at the time when the brain is invaded, the mental symptoms are in the main those which usually accompany an organic insult of a toxic-infectious nature. It is at this time that the lethargy, stupor, coma, delirium or mental confusion stand in the foreground of the clinical picture. In my experience few cases fail to show some degree of confusion or delirium; even when there is a persistent wakefulness, there is, nevertheless, a tendency to delirious dips or phases, especially at night. These may be transient and therefore easily overlooked. Dr. Hunt has just cited a good example of a mild delirium which evidently at the time it occurred attracted little attention.

It is the succeeding neurotic and psychotic manifestations that gradually emerge in the further course of the encephalitis that have been emphasized in psychiatric clinical groupings. Dr. Folsom has shown that a large majority of cases arriving in the state hospital present a clinical picture characterized essentially by an emotional instability plus various behavior disorders and delinquencies. More than 85 per cent of the cases showed this type of reaction and little that went beyond it. I was rather surprised that the more complex psychotic reactions were not more frequently encountered, although paranoid states, schizophrenic syndromes, agitated depressions and manic-like excitements are to be found in the series of cases reviewed.

Dr. Folsom's results seem to support the view that epidemic encephalitis, which pathologically has a special affinity for the brain stem and basal nuclei, has as one of its main clinical expressions a pronounced and often lasting disturbance of the emotional mechanisms. Even in the lethargic or stuporous phases of the disease an emotional disturbance of the nature of a marked inhibition of affect is often the most striking symptom, a fact which has led Kennedy and others to speak of an "emotional stupor." Later in the course of the disease, an opposite tendency is seen in the breaking down of inhibitions and diminished emotional control leading to the behavior disorders which apparently are the most frequently encountered clinical reaction types in hospital cases.

In contrast to the abnormal emotional reaction is the good preservation of the higher psychic functions — the intellectual faculties, memory, orientation and mental grasp are usually quite well preserved. The absence of deterioration or dementia, such as accompany other organic brain diseases, is an outstanding fact even in advanced cases of encephalitis. One frequently observes patients who appear quite dilapidated, careless of their persons, inactive, dull and with a vacant expression and the usual parkinsonian postures. However, on examination one finds that such patients have a good grasp of the surroundings, well preserved memory and ability to give correct information about themselves and their sickness, showing that there is little or no mental deterioration.

Dr. Folsom, in speaking of the relation of preceding personality types to mental symptoms, touched briefly on a point about which there has been much discussion; namely, what is the explanation for the more complex psychotic clinical pictures that sometimes appear in the course of an encephalitis — such as agitated depressions, paranoid states or schizophrenic syndromes? It could hardly be maintained that postmortem findings help in understanding these complex psychotic reactions. They seem to be much more closely related to the underlying personality make-up and special life experiences of the individual. Certainly, in other organic conditions it has been shown that mental symptomatology is to a large extent conditioned by the underlying personality and psychologic make-up of the individual. Dr. Folsom gave examples of this relationship in two of the cases which he briefly described. One was the case

of a boy who showed very odd behavior which, on analysis by Dr. Hinsie, was shown to be definitely related to some previous experience and submerged wishes of a sexual nature. These were quite obviously expressed and realized with little or no symbolism in the attitudes and motor reactions which he exhibited during the psychosis. The other case, the one exhibiting a schizophrenic syndrome, had developed on a definite schizoid background. One could almost feel that this was the only type of reaction that that particular person would be capable of showing in the presence of an organic brain disease. The same principle applies, I think, in general to the more complex psychotic reactions. They are apparently best explained, so far as the symptomatology goes, on the basis of the psychology of the individual.

Dr. Bernard Sachs: I am afraid the average person would say we have been making altogether too much fuss about epidemic encephalitis, but the historian of the progress of neurology will have to say that there was a distinct awakening at the end of the Great War with the coming of epidemic encephalitis and with the incident publications and researches on extrapyramidal disease. The effect that this one disease has had on neurology of the present is very remarkable. Nothing has happened to us or to the community that can be compared with the importance of this disease, for it has had its effect on almost every branch of neurology, and particularly on the diagnostic powers of neurologists.

As for some of the special questions that have come up, I shall refer to some of the things I think are of practical importance. Dr. Kennedy is very ingenious in many of his statements regarding the development of the neuroses and psychoneuroses. I see only one danger. After a person has had a syphilitic infection, everything that occurs is attributed to the syphilis, and that is not always correct. Just so, not all mental changes are necessarily sequelae of the disease, although I believe a great many of them do follow it. I am not so much surprised by the number of neuroses and psychoneuroses as I am by the paucity of them. Considering that the disease begins with what is a distinct psychic symptom, the lethargy, for instance, it is astonishing that the rest of the symptoms point to purely somatic changes, and are not distinctly psychic; but that there should be a marked development of psychoses after the disease is not at all astonishing. If we bear just two facts in mind in regard to the disease we will be able to explain many of the things that seem rather anomalous. First, it is distinctly a toxic infection. I do not wish to draw too sharp a line between what is an infectious disease and what is a toxic disease. Looking at it as a toxic disease, there is nothing surprising in the fact that there are in its wake conditions that resemble the ordinary toxic psychoses. You will find that after encephalitis, when psychoses are developed, they assume a variety of different forms. The next point to remember is very characteristic of this infection; that it happens to strike the extrapyramidal system much more intensely than any other part of the nervous system, so we have the peculiar association of the special symptoms in these cases, together with the rather considerable development of psychoses.

So far as the third paper is concerned, I would like to speak of the "unusual signs." The contribution was very interesting, but if any one were to ask me what is the striking outcome of our experience with epidemic encephalitis, I would say that we shall have to revise our conception of the morbid changes in some apoplectic seizures. That has not been brought out here, but it is a point of practical importance. There was a time when we saw people of middle age who were stricken with what would appear to be an ordinary

hemiplegia and for which there seemed to be no sufficient reason. Under ordinary circumstances we would have made the diagnosis of hemiplegia due to thrombosis, because as a rule the attack came on without loss of consciousness, with a gradual development of the hemiplegia. The peculiar fact is that some of these cases have cleared up with unusual rapidity — and I think I am stressing an important point in differential diagnosis — leaving scarcely a residuum of the paralysis. That is not the ordinary experience in hemiplegia due to a thrombus, so I believe that encephalitis is much more common as a cause of apoplectic disease than it is supposed to be.

Attention is called to a fact that is not often referred to, which Oppenheim, one of the best neurologic diagnosticians of our generation, cited many years ago as a typical symptom of encephalitis that there is an aphasia associated with a form of hemiplegia, and a rapid recovery of that aphasia. He recognized that as a symptom of encephalitis. In the majority of cases recovery is quicker than if it were due to a thrombosis. Again probably all of you know of people, of any age, who walk into your office with double vision; they are the people whom one suspects of being in the first stages of an encephalitis.

Dr. Smith Ely Jelliffe: A few suggestions come to my mind that possibly may be of some service. First, von Economo is very well. I saw him this summer. He was then a gentleman farmer at his castle, and we traveled over his farm, looked at his pigs and his chickens, and talked about encephalitis. He expressed great interest in the great extensions which had been made to his studies. He had gone straight on with his scientific work, and had just completed an extremely interesting work for neurologists, concerning which you will all know probably from the publishers. It is a large, complete and important atlas of the nervous system, practically a vade mecum of the entire nervous system, with every cell in it photographed. Thus we can hope to get to that point to which Dr. Kennedy, in a somewhat illusory manner, attempts to lead us; namely, when we can put our finger on the soma, and say, "There is the disease," possibly forgetting that the secrets of function are not seeable in structure.

The important thing that we have learned in this encephalitis epidemic, and Dr. Sachs partly indicated it, has been that we have come more to appreciate how intricate and complex is this mosaic of the human being. We have, finally perhaps, learned that the human machine is a million years old and more. It is so full of complexities that we will never analyze it; but encephalitis has helped us enormously in this differentiating process. Marie, in his charming introduction to Gabrielle Levy's fine study on encephalitis, spoke of the virus which, exploding like a shrapnel shell, involved all parts of the nervous system. Our study of the series of shrapnel-like explosions has influenced the subject of neurology much and has made encephalitis such an interesting problem for study.

There is no use talking about the soma and the psyche as things separate. They were born together; one is as old as the other, and they cannot be separated. What we are interested in is why did that boy drink so much water; why the dyspnea; why did he have polyuria, etc.? We are interested in the functional behavior disturbances of the man. We cannot say that he had a dyspnea solely because of an interference in the respiratory tracts. We know that is not so. We know that the respiratory difficulty was more than a somatic expression of pathway blocking; there was also the effort on the part of the person to express some purpose through his behavior. What this latter was no analysis of the soma is ever going to tell us, but analysis

of the individual's personality may throw some light on it; and in that sense I agree with Dr. Kirby and Dr. Hunt that unless we make a study of the personality we shall not understand these mechanisms.

I think Dr. Sachs was very acute in what he said about being surprised to find so few neuroses in these patients. Why? We see all kinds of so-called neurotic symptoms in a variety of pathologic syndromes, but any one who is a student of neuroses per se does not confuse them with the heterogeneous irregular grouping of neurotic symptoms seen in these encephalitic cases. He knows that a neurosis is more of an entity. A series of behavior disturbances is not to be called a neurosis or psychoneurosis simply because of a combination of so-called neurotic symptoms, at least not in the past twenty years. There has to be some cohesion in the genesis of the behavioristic reaction to entitle it to come within the recognized classes of neuroses and psychoses. Thus I feel that Dr. Kennedy's emphasis is slightly misplaced when he says we must pay more attention to the soma and less to the analysis of the personality. We can influence them a great deal by an analysis of their symptoms from the genetic standpoint, rather than by being satisfied in localizing the somatic lesion, interesting though that certainly is.

I had the pleasant and unusual experience to find at the Deutsche Psychiatrische Verein in Cassel that Dr. Stern, who has written a large monograph on encephalitis, had read a paper of mine on the subject, and wanted to know more about the patient. He asked me what happened to the patient I had reported in the *Journal of Nervous and Mental Disease*, June, 1925. It was a case of encephalitis, with dyspnea, parkinsonism and all the signs that Dr. Kennedy reported in his case. I said that he had made an excellent recovery, and he asked me to tell him how a patient with an "organic process" like encephalitis could get well. I said, "I analyzed him." I attempted to tell him, but I shall not burden you here, beyond just this one detail. You all know that function is going to operate at the easiest level; this is a general economic law. The simplest form of response to a stimulation is the ordinary spinal reflex. A next higher is a more complex reflex, and the higher we go, the more complex the reflex arc activities, until we reach that mechanism for which Freud has used the word sublimation, which represents the highest form of reflex activity to meet life's situations. Encephalitis hits a good deal of the cortex as well as the basal ganglia. It impinges a good deal on the mechanisms of the super-ego. The person is thus prone to short-circuit his efforts in trying to get his instinctive cravings over, but his super-ego has not entirely gone. The intelligence remains more or less intact, as Dr. Kirby has accented. The super-ego remains able to cope with the situation more or less, and what he does is to distort his wish activities into some aberrant or disguised symbolic forms. The dyspnea, polyuria, etc., are wish-fulfilling mechanisms at lower levels, not entirely withdrawn from censorship because the super-ego has not broken down.

With regard to what Dr. Hunt said about reinfection, I agree about the chronicity of the infecting material, but I would emphasize the fact that the psychologic mechanism of regression may be as important a factor as reinfection. By analogy, we know that a person who has a broken arm or a broken leg turns to the easiest way of doing things, and would lock up his arm or leg by disuse. We have to use physiotherapy, etc., to make the person utilize an organ which, because of such inaction, has become below par. In the same way, then, we have to take hold of the encephalitic patient from the aggressive side and make him use the organ, his super-ego, which is below par, and if

we can once force the stimuli over the pathways, which were partly involved, we are approaching cure, and reinfection seems to be arrested.

DR. GRIMBERG: With regard to educating a patient who is suffering from a postencephalitic syndrome, we should not forget that we are dealing with a lesion of the extrapyramidal system; the slowness of motion and abnormal attitude are due to that lesion. You cannot make impulses for voluntary motions run through that system, because they do not, their route is through the pyramidal system, which in such patients is not destroyed. The improvement noticed in such patients, by analysis or otherwise, is probably a natural remission, such as is frequently seen, and should not be conceived as an improvement at all. Of course, psychanalysis may greatly influence a neurosis following an encephalitis, just as it would influence a neurosis following any infectious disease.

DR. KENNEDY: I have attempted in my paper to remarry Soma and Psyche —their divorce had been promulgated by the psychanalysts, who had further cast Soma into outer darkness whence it seemed profitable to recall him.

THE CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 19, 1925

SANGER BROWN, *President, in the Chair*

FACIAL DIPLEGIA IN NEO-ARSPHENAMINE POLYNEURITIS. G. B. HASSIN and CHARLES F. READ.

Multiple nerve involvement after treatment with arsphenamine and neo-arsphenamine is rare. Still more rare is a simultaneous involvement of the seventh nerve, while cases showing bilateral involvement of this nerve have not hitherto been recorded. Our patient was a white woman, aged 21, a nurse. May 27, 1924, while in the Cook County Jail, she received an intravenous injection of 0.45 Gm. of neo-arsphenamine. One week later she experienced paresthesias — tingling in the toes and fingers, a feeling of swelling in the upper lip—quiverings and general muscular weakness. In a few days the paresthesias spread to the face and were associated with severe pain. Within two weeks the muscular weakness resulted in generalized flaccid paralysis of all four extremities and the face. June 23, 1924, she was brought to the Cook County Hospital. She said that since the age of 14 her blood had given a positive Wassermann reaction, and that this was also true of her father, a sister and two brothers.

Examination revealed: bilateral seventh nerve paralysis, of peripheral type; complete loss of active movements in the hands, feet, fingers and toes; impairment of movement in the large joints; pain in the muscles, nerves and face; loss of the tendon and of the left abdominal reflexes; impairment of the position and stereognostic senses; hyperesthesia in the upper extremities, anesthesia for touch on the dorsum of the left foot, and electrical degenerative changes in the muscles of the face and extremities. Six weeks after the onset of the illness, marked ataxia was present in the hands with manifest impairment of tactile and pain sensibilities in the upper extremities and feet. The muscle power was slowly returning, although the feet were drooping and slightly inverted and the general musculature appeared flabby and atrophic.

The slight improvement in muscle power was followed by slow, progressive improvement in sensibility, but the pain and the ataxia persisted for six and one-half months after the onset of the illness. One month later the patient was able to walk on crutches.

April 23, 1925, the condition was practically normal, except for asymmetry in the face, which showed remnants of a left facial paralysis — contractures, muscle spasms and associated movements, with a partial reaction of degeneration. The patient was able to resume her nursing duties.

The clinical picture outlined, a generalized neo-arsphenamine polyneuritis with facial diplegia in a person with hereditary syphilis, started with paresthesias, pain and muscular weakness. The symptoms rapidly progressed, resulting in an atrophic flaccid paralysis of all four extremities and the face, scattered anesthesias and ataxia. The condition somewhat resembled a subacute combined cord degeneration. In the latter there is a combined degeneration of the central motor and sensory nerve fibers; in the condition here described the lesion lies in the peripheral nerves. In both morbid conditions the cause of the degeneration is some intoxication, that in subacute cord degeneration being unknown. In our case the neuritis might have been due to syphilis, alcohol or neo-arsphenamine. Syphilitic polyneuritis, however, occurs in the early stages of syphilitic infection, and in the tertiary stage it occurs only in acquired syphilis. Alcoholic polyneuritis does not involve the cranial nerves. Therefore, it must be assumed that the etiologic factor in our case was neo-arsphenamine poisoning. The pathogenesis of neo-arsphenamine polyneuritis is to be sought either in the neurotropic action of the spirochetes, or in the chemical action of the arsenic. The former presumption in our case is hardly justified, for neurotropic action occurs in the early stages of acquired syphilis, while neo-arsphenamine polyneuritis has been observed in patients who never had had syphilis. It resembled the ordinary arsenical polyneuritis produced by arsenous oxide as in the case reported by Raymond and Léjonne (poisoning with Paris green), in which the onset — paresthesias, pain and weakness — occurred eight days, and the paralysis about sixteen days, after the poison was taken. The late onset of the symptoms is the remarkable feature of neo-arsphenamine polyneuritis. In some of the recorded cases the interval was five days, three and even five weeks (Spillman's case). Regardless of the time of onset, the prognosis of neo-arsphenamine polyneuritis is always good, the treatment being purely symptomatic.

INDUSTRIAL PSYCHIATRY. MANDEL SHERMAN.

This paper is concerned chiefly with the problems of adjustment of individuals in industry, and only indirectly with posttraumatic conditions. Industrial psychiatry has developed from the work of industrial psychologists during the World War, and of psychologists engaged in army personnel work. The influence of mental hygiene workers has been great, and the general concepts of psychanalysts have been important factors in stimulating psychiatrists to enter industrial problems.

The methods of study and classification of personality types used by industrial psychiatrists are many, and a few only can be touched on here. Several systems have been based on psychologic theories. Pruette and Fryer distinguish two personality types: the repressed, and the elated. The maladjustments that result are studied on the basis of these reaction types. Another method of personality classification is that based on changes in instinctive reactions. The psychiatric classifications have been rather loosely constructed.

One system in use until recently makes three groups: the egocentric, the inadequate and the emotionally unstable. This classification is open to serious criticism in that any person may properly be classified in any one or all of the three groups; the usefulness of the grouping consequently resolves itself into the presence of "more or less" of one or the other of these characteristics. Psychanalysts have attempted to study industrial problems, especially those which result from maladjustments due to improper vocational selection, on the basis of identifying the conflicts which the person had developed early in life and which resulted in the selection of a particular vocation. For example, a worker may become unadjusted because he selected his vocation for its similarity to that of his father. Such a choice may be due entirely to identification with the father. Other persons, in order to differentiate themselves from their fathers, may choose some vocation totally unlike that of the father.

An attempt has also been made by Pratt to study industrial maladjustments on the basis of diagnostic groups. He classifies four types that make up the bulk of industrial misfits. These are the feebleminded, those who are "superior" to the job, the "psychopaths" and the "psychotics." A method of study which is rather new in industrial psychiatry has been attempted by Elton May. He studies the workers on the job, attempting to discover the dominant reveries of each individual. He is not interested primarily in conflicts developed in the past life of the person, but tries to find the significant discrepancies between his reveries and the demands of the work. May's work has produced interesting and rather significant results.

Analysis of the theories and methods of industrial psychiatrists shows two factors which are detrimental to the scientific advance of the problems involved. First, the work of the psychiatrist may be roughly termed individualistic; one observer may use certain methods, terms and classifications which another observer may not be able to understand. This difficulty arises because many psychiatrists think that classifications may be made at will, justifying the particular methods they use by the resulting cures. Often the cure is not caused by the method of therapy at all, but results from social readjustments, change of environment in general, the patient's own recognition of his difficulty and so on. The second detrimental factor is the persistence of the psychiatrist in using almost entirely subjective methods of study. The only method he employs, in the last analysis, is that of the interview. Such procedure is not only open to many errors, but is inadequate in that it does not lend itself well to teaching purposes, and the data secured are usually of doubtful value in research.

DISCUSSION

DR. L. L. THURSTONE (by invitation): I wish to endorse everything Dr. Sherman has said. I find myself entirely in sympathy with the spirit of his discussion of industrial psychiatry and of the conditions that should favor scientific advance in this subject. During the last six or seven years I have had the opportunity to see experiments in personnel management carried out in New York, Pittsburgh and other places. Some of these projects I have been privileged to observe at close range, and in some of them I have personally assisted. It has occurred to me that personnel management might be defined as the attempt to coordinate in some systematic fashion the two realms of working and living. The successful personnel manager is characteristically the man who not only understands thoroughly the effect of living on work as well as the effect of work on living, but who also attempts in a systematic way to bring about conditions which will favor both. It is clear immediately

that such a task is also part of the function of industrial psychiatry. In fact, it seems that there are at least four professional groups claiming certain phases of this task as peculiarly their own. These professional groups are represented by the personnel manager, the educator, the psychiatrist and the psychologist. Even the theologian lays claim to a part in this work.

Obviously, individual problems may arise that make it difficult to determine which of these professional representatives should be asked for advice. Certainly the services of the psychiatrist would hardly be needed to adjust minor difficulties which could be handled by mature and sensible people on a friendly basis. Possibly the personnel manager should be expected to handle such problems of minor personnel adjustment in an industrial plant. When the personnel trouble is a matter of training or education, the educator should be a useful advisor. For handling problems of selection and placement, the psychologist has developed an objective technic which is gradually invading the field of personnel management. The psychiatrist, in his capacity as a medical expert, should be appealed to only in those difficult cases which call for medical treatment. The psychiatrist who decides to devote himself to personnel management would, of course, be an exception. In such a capacity he is working not as a medical man primarly, but as an industrial personnel manager, and in that capacity he may find his psychiatric experience of great help. It is certainly highly desirable for educators, psychologists, personnel men, and theologians to learn enough of psychiatric methods to be able to handle the simpler maladjustments in an enlightened way and without bothering the expert psychiatrist. Especially difficult cases, requiring consultation with the psychiatrist, would thus be detected early.

Dr. Sherman mentioned the problem of classification in psychiatric work. In our attitude toward classification in psychiatry we may adopt either one of two extremes. We may take the advice of Dr. Adolf Meyer and others so literally that we refuse to acknowledge that there should ever be any classifications, or categories, or groups in psychiatry. We shall then be working with individuals only, without recognizing that individuals fall into types. As a matter of fact, that is not only humanly impossible, but it would also be highly undesirable, even if we could take literally such an ideal. On the other hand, we might become so concerned with classification that we should be completely satisfied as soon as an individual or a patient had been properly pigeonholed. It is quite evident that progress in helping people cannot rest solely on classification as its main objective. Progress in the art and science of psychiatry, in its industrial as well as in its other aspects, can come only so far as we find similarities and differences among cases, and in the extent to which we discover, in handling various types and classes of cases, that certain methods are successful or unsuccessful. No practical technic can be developed in any field, and no scientific generalizations can be made, unless we are willing to classify subjects by study of their similarities and differences. Human beings constitute no exception to this guiding principle of research. Classification is not only laudable but also indispensable so far as it serves as a means to an end. But it ruins the usefulness of a profession if it becomes itself a final objective.

Dr. Herman M. Adler: I was interested in this discussion and subscribe to everything that has been said. One thing that might be interesting to the society is the work of Dr. J. D. Ball with the Shell Company in California. I saw part of that work and was interested because I think it illustrates one of the solutions of the discrepancy of which we have seen so much. I see no

rivalry between psychology and psychiatry but think there is one distinction between them which comes out in these industrial relations; in the eyes of the layman the psychologist is a highbrow, and perhaps a paid agent of the employer, whose object is to take the worker away from his job and analyze him. About this the worker has nothing to say—he passes or he does not. Whether the psychiatrist is employed by the company or not, he is a physician, and that gives him an advantage with the laborer which the psychologist has not. If he uses this advantage he can get a contact with the laborer much like that of the physician in his office with the patient. He is there to help the man, and the employee can look to him for help. This is what stood out in Ball's work. There is nothing new in his technic; he simply managed to sell his work to the workmen. During the very serious disturbance in the oil fields in California after the war, the Shell Company was the only one that did not have a strike; the company officers believe this was due to Dr. Ball's work. Organized labor there is about the same as elsewhere. There was only one group of men, the detective force, with whom Ball could not get in contact; they were required to bring in so many agitators every week in order to hold their jobs. Dr. Ball had conferences with both employers and employees, and had in mind what was needed for their welfare.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 20, 1925

SHERMAN F. GILPIN, *President, in the Chair*

LEMNISCUS SYMPTOMS FOLLOWING EPIDEMIC ENCEPHALITIS. DR. W. B. CADWALADER.

Two patients were presented who, after having had epidemic encephalitis, developed severe bilateral impairment of the recognition of posture and the recognition of vibration in all four limbs, associated with motor weakness especially of the lower limbs, with increased tendon reflexes and Babinski sign. I attribute these symptoms, which were the only ones present, to a lesion in the median portion of the medulla oblongata in the region supplied by the anterior spinal arteries. This syndrome was first described by Dr. Spiller (J. Nerv. & Ment. Dis. **35**:775, 1908). I know of no cases recorded in the literature in which a necropsy has been obtained. The similarity of these symptoms to those of multiple sclerosis was commented on.

A CASE WITH A QUESTIONABLE DIAGNOSIS OF PARESIS. DR. M. A. BURNS.

This patient is presented because of the peculiar physical signs and symptoms. He is a white man, single, aged 53, with unimportant family and past histories except for an attack of gonorrhea at the age of 21, and a distinct history of alcoholism for a period of from five to ten years. He was well until Jan. 1, 1925, when he had a convulsion while asleep. The attack lasted about twenty minutes, when he again fell asleep; he awoke the next morning feeling fairly well although he did not remember having had a convulsion. After this episode he worked regularly until February 17, when he had a second nocturnal attack, during which he got out of bed and fell to the floor; he

does not remember anything about this attack but was told that it lasted about twenty minutes. April 1, he went out to do some target shooting and while assembling his rifle had a third attack. Immediately after this attack he became confused and remained so for about a half hour. About April 3, he was admitted to the University Hospital at Ann Arbor, Mich., where he remained for three days. During this stay at the hospital, examinations of his blood, spinal fluid, urine and eyes, and roentgenograms of his head and back, gave negative results.

He came to Philadelphia about May 15, and worked steadily until May 25, when at night while asleep he had an attack and on the following day had a spell on the street during which he fell and became unconscious. August 1, he had an attack, and several days later had another attack during the day. In the last two spells he had incontinence of urine. From Jan. 1 until Sept. 10, 1925, he had thirteen convulsive seizures associated with unconsciousness — four during sleep and nine diurnal. He has not had an attack since September 10.

He was admitted to Jefferson Hospital, Philadelphia, September 16, and left September 24. Two spinal fluid and two blood examinations were made and all showed negative Wassermann reactions. One spinal fluid examination showed four cells and the other none.

Examination.—A fine tremor of the hands, lips and tongue is easily noted. The pupils are irregular and unequal, and do not react to light; the eyegrounds show slight sclerotic changes. The reflexes are present. The patient's memory for recent events is not as good as it was some years ago. He has a feeling of well being except for the thought of his convulsive seizures and does not seem to be especially worried about his condition.

DISCUSSION

DR. EDWARD A. STRECKER: I would like to know if any one has ever seen a case of paresis proved by necropsy, in which there was a negative serology during life.

A CASE OF MENINGIOMA OF THE RIGHT FRONTAL REGION. DR. CLARENCE A. PATTEN.

This case is presented because of several points of interest: the history of violent headaches over a period of from twenty to thirty years, and the question whether these headaches could have been due to tumor; the mental symptoms which developed during the recent and most rapid growth of the tumor, possibly pointing to frontal lobe involvement; the binasal hemianopia occurring with a tumor in the right frontal region; the absence of all neurologic findings except those of the second nerve and the weakness of the lower left face, the latter being considered the only reliable evidence of a lesion in the right frontal lobe; the roentgenologic confirmation of tumor of the right frontal region; the restoration of vision after such a long period of papilledema.

The family history is practically of no importance. The patient was 52 years of age, was a barber by trade and used alcohol moderately. He had been married many years without issue, which may have been due to a gonorrheal infection in early life. He had been obliged to wear glasses for working and reading for about thirty years, and during this time the glasses had been changed at least eight times. The headaches which he had had for about

thirty years were attributed to eye strain as they always came on Sundays following long hours of work on Saturdays. He described the headaches as being so severe that he felt like banging his head on the table or against the wall. They were only slightly relieved by bromide preparations and acetylsalicylic acid, both of which he took in large quantities.

He gave up his position as a barber two years before he entered the hospital (May 15, 1925), giving as a reason that it had become difficult for him to see well enough to do the close work required. He accepted a position in an electrical supply house handling supplies. As time went on it became necessary for him to get his fellow workers to punch the clock for him as he could not see well enough to do it himself.

In May, 1923, he first showed nervousness; his hands would shake if he was excited, teased or irritated. Sometimes he had attacks which he described as "feeling like stage fright." Early in 1924, he began to feel drowsy and slept more than formerly. He noticed a slight difficulty in walking, and thinking this might be due to some trouble with his legs, ordered elastic stockings. His wife noticed that he was becoming less particular in the matters of his dress and eating. All symptoms became progressively worse and in January, 1925, they were very marked. He fell asleep at the table; lost interest in everything; had to be urged to go out or to dress himself. Later he became lost in his own house, apparently having no memory for the location of the doors. Often he would think he was in the bath room and urinate on the floor wherever he happened to be. Many times when in bed he would openly masturbate. He ate his food with his fingers. At times he sang for long periods without interruption, and whereas formerly he had been very witty and jolly, he had now become actually silly. Of all these symptoms the patient at present has no recollection.

When the patient entered the hospital the only findings were the binasal hemianopia and choking of the disks of about 3 diopters. So far as getting around the ward was concerned, he was practically blind. He had no headaches and no vomiting. There were no symptoms referable to the viscera. After he had been in the hospital two or three weeks, it was noticed that the lower part of the left side of the face was distinctly weak, and there appeared to be some uncertainty in the use of the left arm. The choking of the disks increased rapidly and at the time of his transfer for operation was about 7 diopters. There were old and new hemorrhages in each retina. A frontal lobe tumor was suspected. A sugar tolerance test gave the following findings: fasting specimen, 118 mg. per hundred cubic centimeters of whole blood; one half hour later 170 mg.; one hour later 163 mg., and two hours later 158 mg. There was no urinary sugar. Roentgenograms of the sella turcica showed a clear outline, with the dimensions of 1 cm. anteroposteriorly and 1 cm. in depth. A few calcareous bodies were present. It was thought that the sugar curve indicated pressure on the pituitary body and that the lesion was not in the pituitary gland itself. Spinal fluid examination gave negative findings in all respects, and the blood Wassermann reaction was negative. The urinary findings were consistently negative and the blood chemistry showed no evidence of renal disease.

A roentgenogram of the entire skull showed a definite area of opacity in the frontoparietal region about 4 inches (10.16 cm.) in length, due to thickening of the bone with marked increase in density. It was a sharply localized lesion and did not conform with the general thickness of the skull. It was regarded as pathologic since there seemed to be areas of erosion and irregularity on

the under surface. It suggested some type of tumor. The posterior clinoid processes seemed to be very thin; this might have been due to erosion.

The patient was removed to the University Hospital, where he was operated on by Dr. Francis C. Grant, a large endothelioma being removed from the right frontal lobe near the median line. The details of the operation are to be reported by Dr. Grant in a subsequent paper. Three weeks after the operation the patient was able to read words in large type and since that time, now about four months, vision has been greatly restored. The last report (by Dr. B. F. Baer, Jr.) of vision is: right eye, 6/9 and left eye, 6/15. The visual fields still show binasal hemianopia with marked contraction of the temporal fields.

This is unquestionably a case of endothelioma which, following Cushing's classification in 1922, may be subclassified as a meningioma. An excellent summary of the roentgenologic aspects of meningioma is given by Drs. M. S. Sosman and T. J. Putman (Am. J. Roentgenol. **13**:1 [Jan.] 1925) from the Peter Bent Brigham Hospital, Boston.

DISCUSSION

DR. CHARLES K. MILLS: The first tumor distinctly revealed by the roentgen ray was reported by Dr. Archibald Church, and some reference to the paper of Dr. Church should be made in Dr. Patten's report. The second one was shown by Dr. Pfahler in a case of my own. This was a tumor of the parietal lobe and it was partially removed by operation. The case is on record and it is the one to which Dr. Patten referred. It is quite true that disorientation phenomena may be present in frontal lesions, but it is equally true that disorientation phenomena may result from bilateral lesions in more posterior regions of the brain. Dr. Rhein reported one such case, and I remember reporting another seen by me in consultation with a physician living outside Philadelphia. I believe there is an orientation center in Flechsig's posterior association area.

DR. WILLIAMS B. CADWALADER: Dr. Patten said his patient had had difficulty in finding his way about his own house and had, as he expressed it, "gotten lost." Judging from this description the condition may have been a form of disorientation in space resembling that described by Marie in Paris just after the war. He observed this condition in soldiers who had had extensive destruction of the frontal lobes of the brain from shell wounds. I have been watching for this symtom, but have not observed it in a few cases in which there was a unilateral frontal lobe lesion of moderate size. It may be that an extensive bilateral lesion is required to produce the condition and I think Marie stated this. In Dr. Patten's case both frontal lobes seemed to have been severely affected by compression.

DR. WALTER FREEMAN: Until the time of Dr. Cushing's report on the meningiomas, thickening of the bone overlying these tumors was looked on more or less as a curiosity. Dr. Cushing, or maybe it was a German writer, showed that nearly 50 per cent of patients with meningeal tumors showed thickening of the bone sufficient to be noticeable in the roentgenogram. Thickening of the bone, however, is not a positive indication that there is a tumor beneath. We have found skulls at necropsy, for instance, that showed marked local thickening, particularly in the frontal area, usually associated with pachymeningitis. I also remember a soldier at Camp Dix who was hit by a flying stove-lid which produced a compound fracture through the frontal sinus.

During the ensuing months marked exostoses developed in the region of the fracture, with the formation of a spicule of bone 5 cm. long that penetrated between the cerebral hemispheres.

In regard to the loss of the sense of direction, experimental physiologists working on the frontal lobes have been struck by this phenomenon. I refer particularly to Bianchi's work on dogs and monkeys. After extirpation of the left frontal lobe, and more markedly after extirpation of both lobes, these animals lost the ability to find their way to their kennels. I have noticed the same phenomenon in a colored man who presented three separate gliomas in the left frontal lobe.

Dr. Clarence A. Patten: There is something characteristic about the bony changes of the skull in these meningiomas which differentiates them from general thickness of the skull. Dr. Sosman and Dr. Putman mention spicules and honeycombed appearance of the bones, and they emphasize the point that the tumor itself does not need to be widely attached to the calvarium. From the pathologic standpoint it is evident that the tumor cells attack the bone, producing these distinct changes. In regard to the spatial disorientation which was mentioned in the discussion, I have considered that it was impossible to eliminate the factor of defective vision and the general clouding of consciousness. These, I believe, account for the patient getting lost in his own home, and the spatial disorientation, as reported by other writers, did not exist in this case. Another interesting point in this case is the pathologic sleepiness, which could not in any respect be attributed to a general increase of intracranial tension.

Narcolepsy Occasionally a Postencephalitic Syndrome. Dr. William G. Spiller.

Narcolepsy in its typical characteristics has usually been regarded as rare, but I have had three cases within the past year. These cases were described in detail. There seems to be some relation to encephalitis. A few cases of narcolepsy with a close association with encephalitis have been reported in the literature. There is a tendency to consider narcolepsy merely as excessive sleeping, even at inopportune times, but the sudden weakness which develops on emotional disturbance or excitement, sufficient in some instances to cause the patient to fall, is also a part of the syndrome.

Encephalitis with Monoplegia. Dr. B. F. Alpers.

The case of a man, aged 45, an electric welder, is presented as one of encephalitis with a monoplegia or monoparesis. The diagnosis, while not absolutely established, seems the best one possible in this case although other possibilities must be considered. The patient entered the Philadelphia Orthopedic Hospital in June, 1925, and was discharged in September of the same year; his complaint was that he had been unable to move his right arm for four days. He had worked as an electric welder in both this country and Canada, but not in a way which involved undue use of his right hand. His task consisted of welding together pieces of iron, most of the work being done by machine. The marital, family and past medical histories were unimportant.

He was well until two or three weeks before entrance to the hospital, when he noticed that he could not close his right hand; this lasted only about from three to five minutes. He resumed work immediately afterward. For two or three weeks this happened every day, but since May 30, 1925, he has

been unable to close his right hand at all and his right arm feels very weak. He has had no pain in the arm or trouble with his right leg. At times he has noticed that he could not say what he wanted to say and occasionally that it was hard for him to speak at all. He said he had seen double, this apparently having occurred some time before the onset of trouble with his hand.

Physically, the patient is fairly well developed. When first seen he was slow at answering questions, though he comprehended them readily. All his teeth were carious and he had marked pyorrhea. The teeth were removed. The heart and lungs were negative. The cranial nerve examination showed irregular pupils reacting sluggishly to light, and a left palpebral fissure narrower than the right. There was a slight but definite weakness of the left face. A definite weakness of the right hand was noticed, the dynamometer reading with the right hand being 0, and with the left hand 100. The patient was unable to flex the fingers of the right hand; the hand was held in partial wrist drop with the fingers semiflexed. Movements at the wrist joint were limited in flexion and extension, but motion and power were good in the elbow and shoulder joints. Occasionally there was a clonic movement of the thumb and forefinger of the right hand. No tenderness on pressure over the nerve trunks was elicited. The arm reflexes were more active on the right than on the left, and both knee jerks were very active, though more so on the right. There was no Babinski sign.

The blood picture was entirely negative. The spinal fluid showed 2 cells, and negative globulin and Wassermann tests. The colloidal gold test showed some reduction in the second zone. Electric examinations were negative.

The progress of the case is of particular interest. It was learned that the patient one and one-half years previously had got a small piece of iron into his right hand, and this showed with the roentgen rays. It was not in a position to cause symptoms, being in the web between the thumb and forefinger. During his stay in the hospital the patient lost his power of speech entirely and this has never returned completely, though it has improved slightly. At the same time he became much more stupid and his attitude more facetious, and this has persisted without change up to the present. He understands questions but cannot express himself, and he is definitely duller, in fact markedly so, than on entrance into the hospital.

Of course, the possibility of a functional disorder must be conceded since the patient was interested in receiving compensation for the foreign body in his hand. It seems hardly possible to me, however, that all the clinical signs presented are explainable on the basis of a functional disorder — the mental deterioration, the pupillary changes, the partial ptosis on the right, the increased reflexes — but I am aware of the fact that my position is not entirely secure, and that questions may arise in the minds of others.

DISCUSSION

DR. CLARENCE A. PATTEN: The diagnostic problem in this case in the beginning was not easy. The fact that there was a compensation element and the striking resemblance to hysteria made us incline to a diagnosis of a functional state. However, after the development of the attack mentioned, the patient had a distinct motor aphasia and a definite mental reduction which, together with the weakness of the right arm, pointed to a lesion of the left frontal lobe. The atrophy of the hand muscles, however, is much more marked than one would expect from a cortical lesion, and we were unable to account for it on the basis of an involvement of the cervical region of the cord.

Book Reviews

SOCIAL ASPECTS OF MENTAL HYGIENE. Addresses by F. E. WILLIAMS, C. M. CAMPBELL, A. MYERSON, A. GESELL, W. E. FERNALD and J. TAFT, with a foreword by J. R. ANGELL, and an introduction by W. R. DAVIE. Cloth. Price, $1.60. Pp. 150. New Haven, Conn.: Yale University Press, 1925.

This series of lectures was prepared in the spring of 1923 for the New Haven Council of Social Agencies with the cooperation of Yale University. The objective, the utilization of the psychiatrist's contributions for the recognition and correction of conduct disorders, was attained so satisfactorily that publication resulted. The social case worker deals with a vast number of psychiatric cases, many of which can be adjusted by her, working at times independently, at times with the cooperation of the psychiatrist. The type of case she is particularly trained and qualified to treat is admirably set forth by Miss Jessie Taft. What kind of training she is to have and how well her problems are covered by such training in psychiatric social work as she may now have in several universities remains to be determined. Certainly she has therapeutic advantages over the psychiatrist when the solution of the social difficulty lies primarily in changes to be made in the environment, and certain analytic advantages when the difficulty is best observed in the milieu. How seriously the worker should be taken by the psychiatrist is considered also by Miss Taft.

Williams, in his lecture on "The Mechanism of Human Behavior," utilizes the evolutionary approach to the problem of emotional disturbances in otherwise competent persons in very successful nontechnical form. The lecture, like the remaining ones in the series, can be recommended to the layman as well as to the social worker, without the usual misgivings one has in placing popularized psychiatry in the hands of a patient. Campbell on "Mental Hygiene and Education" pleads for the study of human behavior as an integral part of the training of the educator, in order to enable him to recognize symptoms of behavior difficulties as they arise in the class room, "to supply the physician with very important data, and to cooperate with the physician in improving the situation in the interest of the development of the pupil."

In "Mental Hygiene and Family Life," Myerson dwells on the housewife's neurasthenia which results from such factors as the sedentary nature of her work, her protest against its monotony, her insufficient convalescence after childbirth or disease, her clash with the husband over the control of the family budget, the fear of childbirth, the ill effects of contraception on sexual satisfaction and the general lack of understanding of courtesy in the sex act. In the mental hygiene of the child he stresses the importance of nutrition and good health habits, of natural development without striving at precocity, of play for its social training, of utilizing praise, reward, blame and punishment, of stimulating without flattering, of hurting without degrading and, finally, of easing off discipline when puberty begins, allowing the child gradually to develop its own power.

Gesell in his lecture on "Mental Hygiene Service for Children" names as four factors in the personality of the child with which mental hygiene is concerned: (1) intelligence and knowledge; (2) "sense of values," a blanket term to cover "adequate appraisal of reality; openmindedness; unwillingness to evade reality; the ability to see failure, evil, truth, justice; an imaginative

sensitiveness to what others are thinking and feeling, particularly about us; the homely virtue of common sense, and the vital spark of mental sanity called the sense of humor"; (3) emotional control, and (4) morale, a term which is "the summation and expression of the other three," their "integration." Illustrative cases are cited in detail. The agencies best adapted to training and conserving mental health are (1) child consultation centers, (2) kindergartens and nurseries, (3) the schools and (4) the juvenile court.

A posthumous essay by Walter E. Fernald explaining in detail the well known Fernald program for the care of the feebleminded completes the series. Fernald shows that only 8 per cent of 3,000 defective public school children diagnosed by the Massachusetts traveling clinic make any trouble; that defectives from good homes are usually well behaved; that adult defectives trained in competent institutions do well after discharge, especially with some supervision; that many defectives never institutionalized are employed at good wages in many industries; that the majority of the adult morons and many of the imbeciles discharged from Waverley have been able to support themselves at good wages, even before the war.

In the progressive states only 10 per cent of the feebleminded are in institutions. A program caring for the rest involves five steps: 1. Identification, that is, examination by psychiatric clinics of all children three or four or more years retarded in school. 2. Registration of all patients diagnosed as feebleminded, with full records. 3. Instruction through special classes emphasizing manual and industrial work, handbooks on methods for school teachers of defective children and tactful manuals for parents. 4. Supervision of the feebleminded through a central government authority directed by a board of control, a state commission for the feebleminded and a state board of health whose responsible officer should be a psychiatrist. Each defective should be regularly visited and kept under observation by a local visitor. 5. Segregation is recommended for the majority of idiots and imbeciles and for those morons whose families are unable to protect or control them. The general aim is to recognize mental deficiency in childhood; train according to capacity; teach correct habits of living, and give friendly guidance throughout life.

LES NERFS EN SCHEMAS. ANATOMIE ET PHYSIOPATHOLOGIE. By A. PITRES and L. TESTUT. Price, 150 francs. Pp. 706. Paris: Librairie Octave Doin, 1925.

This rather unusual book, as expressed in the preface, was born as a result of the war. Apparently the French were confronted with the same problem that worried all of the belligerent nations, in that soon after the outbreak of hostilities they found that functional and organic nervous diseases were far more prevalent than was anticipated. To fill the need of competent neuropsychiatrists the French, like other nations, organized schools of education in neuropsychiatry.

In the Centre of Neurology of the Eighteenth Region, where, during hostilities, almost 12,000 soldiers, either French or foreign, were treated for wounds of the nervous system, the authors periodically instructed physicians in the elements of neuropathology. Confronted with the necessity of teaching them rapidly, they adopted a plan of objective teaching whereby on large pieces of bristol board they drew diagrammatic or semidiagrammatic figures of the nervous system outlining the various centers and tracts in rather startling colors to call attention to them. These diagrams were found of such value that even during the war the authors were urged to reproduce them with the attendant

physiopathologic data in book form, and it was in response to this desire that the book was written. The editing began in 1919 and was finished in 1920, but publication was delayed until 1925.

The book contains forty-one color plates and 164 figures in black and white. The color work was done by Dupret and the charts by Oberlin. The reviewer has never elsewhere seen such beautiful color work in a neurologic volume. A study of the color plates makes clear at once the reason why students were able to grasp the subject quickly and why the authors were urged to reproduce their designs. For example, turning to plate XI, which illustrates the hypoglossus nerve, there is, first, the cortical center of the twelfth nerve in the lowest portion of the precentral convolution. Then there is an illustration of the medulla with the nuclear representation of the hypoglossus and the fibers coming from the medulla, its further course through the bony structure and its distribution to the tongue. Its other supply to the internal carotid, jugular and hyoid bone is shown also in colors, so that the student at once grasps the distribution of the nerve. This scheme is followed throughout for the cranial nerves.

Another plate, plate XV, illustrates the brachial plexus and is equally interesting in its conception. The roots are shown at their exit through the spinal foramina, then the various cords of the brachial plexus, and its branches as they enter the upper arm, which is illustrated by a cross section showing the muscles and bones, there being in the background the chest, clavicle and shoulder, so that the student at once grasps not only the origin of the plexus but also its relation to the shoulder and its continuation in the arm.

Another, plate XVI, illustrating the median nerve, shows the origin in the brachial plexus and its continuation, with a background of the bones of the upper limb, and on the same plate a color illustration of its sensory distribution.

The illustrations of the various tracts in the midbrain, pons and medulla, and of the brain itself are equally clear and striking. This is particularly true of plate XXXVII showing the arterial supply of the brain.

Plate XXV illustrates the relation of the rolandic and sylvian fissures to the external surface, a rather rare illustration in a work on neurology.

The accompanying text is brief but adequate, and fully explains the anatomy and physiology of the structures. It is not at all exhaustive but it is not intended to be. Altogether it is an excellent bit of work and reflects great credit on the authors. It is, perhaps, the best illustrated work on neurology that has grown out of the war.

VAGOTONIA, SYMPATHICOTONIA AND NEUROTONIA. By A. G. GUILLAUME. Pp. 281. Paris: Masson & Cie.

This is the second book by this author on the visceral nervous system; his earlier work was reviewed in the ARCHIVES **5**:3 (Jan.) 1921. The material of the present volume deals essentially with vagotonia as described by Eppinger and Hess (English translation by Walter M. Kraus and Smith Ely Jelliffe, J. Nerv. & Ment. Dis., Monograph Series, no. 20, 1915). The first chapter gives a full description of the material of those authors. The second outlines the theories of the visceral nervous system prior to the work of Eppinger and Hess. The third chapter describes the signs and symptoms of vagotonia and sympathicotonia; the fourth defines the status of these two conditions and what the author calls "neurotonia." This last represents the disorders of both systems, rather than of one predominantly, and implies that one is overactive at the same time that the other is underactive. The next six chapters deal

with purely clinical, physiologic and pharmacologic manifestations and the methods of bringing them to light and describe the appearance of signs and symptoms in various diseases. The final chapter is devoted to diagnosis and treatment.

The book is unfortunate in that it repeats frequently the same general ideas. It contains nothing new, except perhaps the use of the word "neurotonia," which has no great value and merely adds one more word to the present overloaded vocabulary in use in visceral neurology.

The author devotes himself almost exclusively to manifestations of the visceral nervous system in the group of cases which we are in the habit of describing as vagotonia and sympathicotonia, and secondly to those in general medical conditions. The description of visceral nervous signs and symptoms in nervous diseases is of startling shortness, one-half page. More than fifteen years have elapsed since Eppinger and Hess published their monograph, during which there have been collected many data concerning disorders of the visceral nervous system. These data relating to disturbances of the central neurons of the visceral nervous system, of the cell groups in the region of the tuber cinereum, and of their axons, have been of the utmost importance. Epidemic encephalitis has added greatly to our knowledge, yet no account is given of all these facts in Guillaume's book. The third ventricle syndrome is merely alluded to in connection with the pituitary gland, and is not described. War material gave us much information on the effect of cutting the cephalospinal pathways of the visceral nervous system; the work on acute decerebrate states, notably that of Bassett and Penfield, has also added much.

Since the author devotes no attention to the central or cephalospinal neurons, and since he adds nothing to our knowledge of the anatomy, physiology, pharmacology and symptomatology of the peripheral neurons, the book, for the English or German reader, becomes entirely unnecessary, since the former can read the same material in the monographs of Eppinger and Hess, or of Higier (Vegetative Neurology, English translation, J. Nerv. & Ment. Dis., Monograph Series, no. 27, 1919), and the latter can read the material in the original.

The book has a considerable number of illustrations.

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